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Rev. 07/10/02

REMEDIAL COST ANALYSIS

GRANITE CITY LEAD SITE

REACT Environmental Engineers
2208 Welsch Industrial Court
St. Louis, MO 63146

January 11, 1994

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EXECUTIVE SUMMARY

Introduction

The Granite City Lead Site includes areas in Granite City, Madison and Venice, Illinois, where lead contamination has been associated with former secondary lead smelting and lead-recycling activities at the NL Industries/Taracorp Site. The Site includes lead-contaminated soils and waste materials at the main industrial site as well as residential soils in areas adjacent to the industrial property where soil contamination has been attributed to lead fallout from the smelter stack or from battery case material used as fill material in alleys, driveways and other areas.

The Site was listed on the National Priorities List (NPL), 40 C.F.R. Part 300, on June 10, 1986. NL Industries voluntarily entered into an Agreement and Administrative Order by Consent with the U.S. Environmental Protection Agency (USEPA) and the Illinois Environmental Protection Agency (IEPA) in May 1985 to implement a Remedial Investigation and Feasibility Study (RI/FS).

Different alternatives to remediation of the Site were addressed in the resulting Feasibility Study and its Addendum. Subsequent to the Feasibility Study, the USEPA issued a Proposed Plan for the Site in January of 1990 and a Record of Decision (ROD) on March 10, 1990.

In addition to remedial activities associated with the industrial property, the remedy selected in the ROD called for excavation of all residential soils and battery case materials in Granite City, Madison and Venice, Illinois, and any other nearby communities with lead concentrations greater than 500 ppm. The selected remedy called for consolidating all of these excavated materials with the slag pile on the main industrial property (Taracorp pile). The consolidated materials were then to be capped.

This report summarizes and reviews the various cost estimates which have been prepared by Government Agencies for remedial activities in the adjacent residential areas covered by the ROD. The report also includes an independent cost estimate for the remedial activities in the residential areas at the current ROD. The activities associated with remediation of the main industrial property are outside the scope of this report.

Both the Government estimates and this independent estimate are presented on an average per property basis as well as an overall basis. In order to facilitate comparisons to each other and to experiences at similar sites which are also presented in the report, the costs for both the Government and independent estimates are broken out to show those associated with stack-emission contamination (stack-emission properties) versus those resulting from the use of battery case material as fill (remote-fill properties). In the case of the stack-emission properties, the excavated materials were treated as special waste for disposal pricing during the independent cost estimate development. Because of the mixture of special and hazardous waste anticipated at the remote-fill properties, however, disposal options were included in the independent cost estimate. These options included disposal of the hazardous waste as such versus disposal of the hazardous waste as special waste after stabilization.

Summary of Findings

In REACT's review of the Government's estimates, several areas of concern were noted. These included:

1) The lack of consistency in the various estimates presented by the Government for remedial efforts at the Site. Average costs for residential stack-emission properties ranged from $13,900 per property to over $42,000 per property exclusive of inter-Agency management fees which have been attached to work performed to-date. Further, the various estimates peak at the $42,000 range, with later estimates (generated after some of the sites had been remediated) going down to approximately an average of $33,000 per property. This knowledge and experience base appears to be ignored, however, with the latest estimate available from the Government rising back to the $42,000 range.

2) The lack of consistency in the number of properties included in the various Government estimates. The number of stack emission properties discussed ranges from 1300 to 1532. The number of remote-fill properties ranges from 16 to 105.

3) The lack of consistency of the Government estimates on the Granite City Site compared to their experience at other residential remedial sites across the country.

4) The approach used in deciding whether a residential lot should be remediated which may result in entire properties being remediated because of the presence of a small hot spot rather than wide-spread stack emissions.

5) The wide variation/disparity between the total remedial cost estimates obtained by extrapolating the Government's per property averages over the total number of properties to be remediated versus those obtained during the independent analysis. These total project estimates are summarized in the following table.

COST COMPARISON AT COMPLETION
GOVERNMENT-MANAGED VS PRP-MANAGED CLEANUP

Source	Stack-Emission Properties	Remote-Fill Properties	TOTAL*
Government Managed**	$66,639,799	$15,745,663	$82,385,462
PRP-Managed**	$21,374,984	$7,845,016	$29,220,000

* These values do not include smelter site remediation, including the battery-casing pile.
** Assumes a clean-up level of 500 ppm. The estimate for a PRP-managed clean-up at 1,000 ppm is **$15,008,576.**

Details of the derivation of each of these estimates are contained in the following report.

REVIEW OF GOVERNMENT REMEDIAL COST ESTIMATES

Introduction

The available information on the various Government estimates to complete the remedial efforts for the stack emission and remote-fill areas which are a part of the Granite City Lead Site is summarized in chronological order on Table 1. This table includes the estimates leading up to issuance of the ROD--from the Preliminary Feasibility Report through the Proposed Plan for the site and a revised estimate for the selected alternative--as well as two modifications to the ROD. Each of these modifications were issued in the form of an Explanation of Significant Difference (ESD). The first was issued on May 7, 1993, and the second on January 27, 1994. Both of these ESDs called for materials to be disposed of off-site rather than consolidated with the slag pile as called for in the 1990 ROD.

Also included in Table 1 are the March 1993 Pre-Design Field Investigation Report prepared by Woodward Clyde Consultants (WCC) for the USEPA and a November 16, 1993 memo from USEPA's Remedial Project Manager (RPM) declaring this Pre-Design document to be the Remedial Design Document for this site, even though it did not contain any remedial cost information. Although CERCLA does not mandate the inclusion of a cost estimate in the remedial design document, it is customary to do so. This is supported by the RPM's statement in the 1/21/90 "Revised Cost Estimate for Alternative H" (see Table 1) that "more detailed costs will be presented in the remedial design document."

Because the WCC Pre-Design report does not include cost estimates, this report relies on cost information gleaned from a variety of government documents including USEPA memoranda, United States Army Corps of Engineers (USACE) delivery orders for remedial efforts which have been conducted at the site and the 1/21/94 USEPA Explanation of Significant Difference (ESD) discussed above. All of these documents are summarized on Table 1.

As stated previously, in order to facilitate comparisons of the various Government estimates for the Granite City Site to each other as well as to estimates for similar sites and the independent cost estimate presented later in this document, per property remedial costs have been calculated from the available information and presented along with the total project costs in the sections that follow. Further, the cost estimate information is broken down into the two types of adjacent residential area properties included in this review - stack emission properties and remote fill (battery case) properties.

Stack Emission Properties

Cost Estimates: Table 2 presents the available Government per property and total project cost estimate information for stack emission properties.

As Table 2 shows, the original Government estimate appeared in the "Proposed Plan for the NL/Taracorp Site," dated January 10, 1990. This estimate of $13,900,000 was based on an escalation of the costs which appeared in O'Brien and Gere's "Preliminary Feasibility Report" of August 25, 1989. The Agency's escalation was performed to account for their lowering of

TABLE 1
SUMMARY OF GOVERNMENTAL REMEDIAL COST ESTIMATES
GRANITE CITY LEAD SITE

REPORT	DATE	COMMENTS
Preliminary Feasibility Report (O'Brien & Gere for NL)	8/25/89	Selected Alternative C: Remediation of "Areas" 2 and 3 to 3 inches. Action level of 1000 ppm in residential areas. Cost estimate: $6.4 million
Proposed Plan for the NL Industries/Taracorp Site Granite City, IL (USEPA)	1/10/90	Selected new Alternative H: Remediation of "Areas" 2 - 8 to 6 inches. **Residential action level of 500 ppm.** **Cost estimate: $13.9 million (Cost arrived at by EPA by escalating O'Brien & Gere's 1989 estimate)**
Revised cost estimate for Alternative H (USEPA)	1/21/90	Corrected 1/10/90 errors. Cost estimate: $21.3 million 1532 properties included. Correlates to average of $13,900/lot.
Revised Final Scope of Work for Rapid Response, NL Industries/Tara Corporation (USACE)	1/27/93	Attachment to Delivery Order 0058, Contract DACW45-90-D-0516 from USACE to OHM Corporation, allocating $3,200,350 for remediation of 17 properties designated as "remote fill" areas (Average of approximately $190,000/property)
Modifications to D.O. 0058 (USACE)	7/8/93- 11/12/93	**Added 23 remote fill areas at average of $130,000/property and 33 residential (special waste) lots at an average of $42,800/lot** to the scope of work, bringing total authorizations to OHM Corporation to $7,735,440. **(Exclusive of USACE mark-ups)**
Explanation of Significant Differences (USEPA)	5/7/93	Modified the ROD to require that excavated battery case material with lead concentrations greater than 500 ppm, which pass the TCLP test be disposed of at an off-site landfill. No cost data included.
Pre-Design Field Investigation Report (WCC for USEPA)	3/93	Characterization data only. No costs included.

TABLE 1 (Continued)

REPORT	DATE	COMMENTS
Remedial Design for the NL Industries/Taracorp Site in Granite City, Illinois (USEPA)	11/16/93	Memo issued by USEPA's Remedial Project Manager declaring the September 1993 Pre-Design Field Investigation Report (see above) to be the Remedial Design Document for the site.
Preliminary Estimate of Additional Cost of Off-Site Landfilling of Excavated Residential Soil (USACOE)	12/93	Estimate of "initial" $2,100,000 additional cost to landfill residential soils rather than consolidating them with Taracorp pile. Reflects total project cost for remediation of 1300 stack emission properties (See listing below).
Explanation of Significant Differences (USEPA)	1/27/94	Allows wastes from residential "stack emissions" to be disposed of off-site as "special waste" rather than being consolidated with Taracorp pile. Early version obtained from Granite City Library contained revised cost estimate. Pertains to residential soils >500 ppm only. No hazardous waste disposal included. Estimate based on experience at site. **1300 properties** included in estimate. Cost estimate: $42,458,410 (or **$32,660/lot**) **Note: Includes USACE mark-ups.**
Delivery Order No. 17, Contract No. DACW-45-89-D-0506, USACE to OHM Corporation	7/19/94	Allocates $3,008,547 for remediation of 70 residential sites. This corresponds to an average of **$42,979 per property**. **Note: Does not include any USACE mark-ups for management of contract as included in ESD estimate of 1/27/94 (above).**

NOTE: Remedial Project Manager, in verbal communication, reports that no estimate has been prepared for remediation of battery-chip areas. Further, no estimate will be prepared until ROD is reopened and a decision is reached regarding remedial activity for these areas.

TABLE 2
SUMMARY OF GOVERNMENT COST ESTIMATES
STACK EMISSION PROPERTIES

Source of Estimate	Average Cost per Property	Estimated Cost at Completion
Original	$13,900/site[1,2]	$21,300,000[1,2]
D.O. 0058, Modification P00005	$42,800/site[2,3] ($46,866 with USACE mark-ups)	$60,604,800[4] ($66,362,256 w/ USACE mark-ups)
USEPA ESD dated 1/94	$32,660/site[5,6]	$42,458,410[5,6]
D.O. 0017	$42,979[6] ($47,062 with USACE mark-ups)	$60,858,264[4] ($66,639,799 w/ USACE mark-ups)

Note: All values represent estimates.

[1] From EPA's revised cost estimate for Alternative H dated 1/21/90. Per property value obtained by dividing total of $21.3 million by number of properties included (1,532).
[2] Does not include any USACE mark-ups for project administration.
[3] From OHM Corporation contract with USACE (Contract No. DACW45-89-0-0516). Per property value obtained by dividing total of $1,411, 384 by number of properties included (33).
[4] No value reported by Government for total project. Value obtained by multiplying per property cost by the average number of properties included in Government estimates (1416).
[5] From Explanation of Significant Differences (ESD) obtained from Granite City Library repository. Cost per property value derived by dividing total of $42,458,410 by number of properties included (1,300).
[6] Includes USACE mark-ups on contract fees.
[7] From OHM contract with USACE No. DACW-45-89-D-0506, Delivery Order No. 17.

NOTE: USACE mark-ups of approximately 9.5% over contractor's fees have been added to remedial efforts conducted to-date.

CURRICULUM VITAE

ELLEN J. O'FLAHERTY

Place of Birth: Cincinnati, Ohio

Date of Birth: December 16, 1936

Education:
Ph.D., Chemistry, Yale University, 1964
M.S., Chemistry, Yale University, 1960
A.B., Oberlin College, 1958

EXPERIENCE:

1984-:	Associate Professor of Environmental Health, University of Cincinnati College of Medicine. Director, Toxicology Training Program, 1987-present.
1982-1984:	Research Associate Professor of Environmental Health, University of Cincinnati College of Medicine.
1975-1982:	Assistant Professor of Environmental Health, University of Cincinnati College of Medicine.
1970-75 and 1966-68:	Senior Research Associate in Environmental Health, University of Cincinnati College of Medicine.
1968-70:	On leave of absence.

HONORS:

Phi Beta Kappa
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PROFESSIONAL SOCIETIES AND RELATED ACTIVITIES:

Society of Toxicology. Member of Editorial Board of the official journal of the Society, "Fundamental and Applied Toxicology," 1983-1985.

Associate Editor of the other official journal, "Toxicology and Applied Pharmacology," 1985-1990. Councillor of the Ohio Valley Section of the Society, 1987-present.

Member, Society for Risk Analysis

Member, Society of the Sigma Xi

Member, American Association for the Advancement of Science

Member, American Association of University Professors

Curriculum Vitae
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UNIVERSITY ACTIVITIES:

Member, College of Medicine Grievance Committee, 1985-1987

Secretary, Faculty Forum, 1982-83.

Departmental representative to Faculty Forum Executive Committee, 1983-present.

Departmental representative to College Committee on Graduate Education, 1990-present.

College of Medicine representative to Faculty Senate, 1987-1989.

Member, Degrees Committee of the Department of Environmental Health, University of Cincinnati, 1975-1987; chair, 1985-1987.

Member, Library Committee of the Department of Environmental Health, 1980-1985.

Member, Art Committee of the College of Medicine, 1979-1983.

Member, Commencement Committee of the University of Cincinnati, 1974-77.

RELEVANT SCIENTIFIC ADVISORY AND CONSULTING EXPERIENCE:

Member, Ohio EPA Surface Water Task Force, 1987-present.

Participant in Cincinnati EPA - Department of Environmental Health Cooperative Agreement for criteria document preparation and review, 1987-present.

Author of pharmacokinetic, risk assessment, and criterion formulation sections of a number of EPA water quality criteria documents including Iron and Its Compounds (1981), Dibenzofurans (1981), Endrin (1982), Toxaphene (1983), and the multimedia document Aluminum (1989).

Reviewer or member of external review panel for a variety of other EPA water quality criteria documents including Ammonia (1982), Ethylbenzene (1985), 2,4,5-Trichlorophenoxy propionic acid (1985), and the multimedia document Polycyclic Aromatic Hydrocarbons (1987).

Author of review of 2-ethylhexanol toxicity and contributor to an evaluation of literature on human exposure to 2,4-dichlorophenoxyacetic acid for the 2,4-D industries association, 1986-1987.

Reviewer for water quality criteria document, Perchloroethylene, for California State EPA, 1987.

Consultant to the Lead Industries Association on an ad hoc basis, primarily concerning the kinetics of changes in blood lead concentrations during OSHA-mandated removal of lead workers from workplace situations leading to unacceptable blood lead concentrations.

Curriculum Vitae
Ellen J. O'Flaherty

GRADUATE AND OTHER TEACHING EXPERIENCE:

Kinetics for the Health Sciences - A 3-graduate-credit course covering linear and nonlinear kinetics in single- and multi-compartment systems; acute and chronic exposure; dose-effect and dose-response relationships; physiologically-based pharmacokinetic models; and application of these principles to problems in research and in risk assessment.

Lectures, generally covering kinetics and risk assessment applications, in other graduate courses such as Risk Assessment, Survey of Toxicology, and Fundamentals of Environmental Toxicology.

Course Director for two courses in Continuing Education Program of Department: Risk Assessment: Principles and Applications, and Advanced Risk Assessment: Biological and Environmental Modeling. Lectures in other Continuing Education courses such as Biological Monitoring and the Mini-residency course for physicians.

STUDENT ADVISORY EXPERIENCE:

Advisees:

Michael L. Dourson, Ph.D., 1980.
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Carol F. Silberstein, Ph.D., 1984.
Wayne D. Adams, research advisor.
John D. Hamilton, Ph.D., 1989.
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Michelle D. Andriot, research advisor.
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Dissertation committees for Jon Reid, Michael Dourson, William Thomas, Paul Ringhand (Pharmacy), Gregory Hammer (Pharmacy), Timothy Hardt (Pharmacy), Alan Gerstein (Microbiology), Carol Silberstein, John Buben, Paul Sichak, Orisa Igwe, Mark Toraason, Laureano Leon, Jerry Ann Ward, Joseph Svirbely, Helen Goeden, Deborah Oudiz, Wayne Adams, William Cibulas, Christopher Bevan, Jonathan Meier, John Hamilton, Matthew Himmelstein, Richard Cassidy, Michelle Andriot.

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RESEARCH INTERESTS:

Application of established kinetic principles to new areas; e.g., toxicology rather than pharmacology. Current work: Kinetics of lead transfer among body compartments and its significance relative to lead toxicity. Development and testing of new kinetic approaches to biological questions.

PUBLICATIONS AND PRESENTATIONS:

Books:

O'Flaherty, E.J. (1981). *Toxicants and Drugs: Kinetics and Dynamics*. John Wiley and Sons, Inc.

Book Chapters:

O'Flaherty, E.J. (1987). Modeling: An Introduction. In: *Pharmacokinetics in Risk Assessment, Drinking Water and Health*, Volume 8: pp. 27-35. National Research Council, National Academy Press, Washington, D.C.

O'Flaherty, E.J. (1986). Toxicokinetics and Dose. In: *Proceedings: New Directions on the Extrapolation of Health Risks from Animals to Man*, Volume 2: Toxicokinetics", Chapter 5, pp 5-1 - 5-10. Electric Power Research Institute, Palo Alto, CA 94304. Proceedings of a workshop held November 30-December 2, 1983, San Diego, CA.

O'Flaherty, E.J. (1985). Differences in metabolism at different dose levels. Chapter 3, In: *Toxicological Risk Assessment*, Volume I: Biological and Statistical Criteria, ed. D.B. Clayson, D.R. Krewski, and I.C. Munro, CRC Press, pp. 53-90.

O'Flaherty, E.J. (1985). General principles of toxicology. Chapter 3, In: *Industrial Toxicology - Safety and Health Applications in the Workplace*. Belmont, California, Lifetime Learning Publications, pp. 27-57.

O'Flaherty, E.J. (1984). *Pharmacokinetics*. In: *Risk Assessment and Risk Assessment Methods: The State-of-the-Art*. Division of Policy Research and Analysis, National Science Foundation, Washington, D.C. 20550, pp. 283-320.

Hammond, P.B., Hong, C.D., O'Flaherty, E.J., Lerner, S.I. and Hanenson, I.B. (1982). *The Rat as an Animal Model of Lead Nephropathy*. In: *Nephrotoxic Mechanisms of Drugs and Environmental Toxins*. George Porter, editor, Plenum Publishing Corporation, New York, pp. 267-277.

Bonewitz, R.F., Foulkes, E.C., O'Flaherty, E.J. and Hertzberg, V. (1982). *The Effect of Dexamethasone on the Kinetics of Jejunal Zinc Uptake and Metallo-thionein Synthesis in the Rat*. In: *Biological Roles of Metallothionein*, E. C. Foulkes, editor, Elsevier/North Holland, Inc., New York, pp. 203-214.

Curriculum Vitae
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Papers in Refereed Journals:

O'Flaherty, E.J. (1992). Physiologically-based models for bone-seeking elements. IV. Kinetics of lead disposition in humans. In press, *Toxicol. Appl. Pharmacol.*

O'Flaherty, E.J., Scott, W., Schreiner, C. and Beliles, R.P. A physiologically-based kinetic model of rat and mouse gestation: disposition of a weak acid. *Toxicol. Appl. Pharmacol.*, 112:245-256, 1992.

O'Flaherty, E.J. (1991). Physiologically-based models for bone-seeking elements. I. Rat skeletal and bone growth. *Toxicol. Appl. Pharmacol.*, 111:299-312.

O'Flaherty, E.J. (1991). Physiologically-based models for bone-seeking elements. II. Kinetics of lead disposition in rats. *Toxicol. Appl. Pharmacol.*, 111:313-331.

O'Flaherty, E.J. (1991). Physiologically-based models for bone-seeking elements. III. Human skeletal and bone growth. *Toxicol. Appl. Pharmacol.*, 111:332-341.

O'Flaherty, E.J. (1991). Physiologically based lead kinetics. *Trace Subst. Environ. Health 24*, 44-54.

O'Flaherty, E.J. (1989). Interspecies conversion of kinetically equivalent doses. *Risk Analysis 9*, 587-598.

O'Flaherty, E.J. (1989). Application of pharmacokinetic principles to exposure to chemical mixtures. *Toxicol. Industr. Health 5*, 667-680.

Svirbely, J.E., Singh, S., Pesce, A.J., and O'Flaherty, E.J. (1988). Sulfa-methoxazole and trimethoprim peritoneal barrier transfer pharmacokinetics. *Clinical Pharmacokinetics 16*, 317-325.

O'Flaherty, E.J. (1988). A physiologically based model of skeletal growth in the rat. *Toxicol. Letters 43*, 85-95.

York, R.G., O'Flaherty, E., Scott, W.J. and Shukla, R. (1987). Alteration of effective exposure of dam and embryo to caffeine and its metabolites by treatment with beta naphthoflavone. *Appl. Pharmacol. 88*, 282-293

O'Flaherty, E.J., Adams, W.D., Hammond, P.B. and Taylor, E. (1986). Resistance of the rat to development of lead-induced renal functional deficits. *J. Toxicol. Environ. Hlth. 18*, 61-75.

O'Flaherty, E.J. (1986). The rate of decline of blood lead in lead industry workers during medical removal: The effect of job tenure. *Fund. Appl. Toxicol. 6*, 372-380.

Buben, J.A. and O'Flaherty, E.J. (1985). Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: A dose-effect study. *Toxicol. Appl. Pharmacol. 78*, 105-122.

Sichak, S.P. and O'Flaherty, E.J. (1984). Consideration of the mechanism of pulmonary adenogenesis in urethane-treated Swiss mice. *Toxicol. Appl. Pharmacol. 76*, 397-402.

Curriculum Vitae
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O'Flaherty, E.J. and Sichak, S.P. (1983). The kinetics of urethane elimination in the mouse. *Toxicol. Appl. Pharmacol. 68,* 354-358.

Hirsch, K.S., Wilson, J.G., Scott, W.J. O'Flaherty, E.J. (1983). Acetazolamide teratology and its association with carbonic anhydrase inhibition in the mouse. *Teratogenesis, Carcinogenesis, and Mutagenesis, 3,* 133-144.

Bonewitz, R.F., Foulkes, E.C., O'Flaherty, E.J. and Hertzberg, V.S. (1983). Kinetics of Zn absorption by rat jejunum: Effects of adrenalectomy and dexa-methasone. *Amer. J. Physiology 244,* G314-320.

O'Flaherty, E.J. and Dourson, M.L. (1982). Relationship between urethane dose rate and adenoma latency: relevance of tumor growth rate and target cell number. *J. Natl. Cancer Inst. 69,* 859-865.

Dourson, M.L. and O'Flaherty, E.J. (1982). Relationship of lung adenoma prevalence and growth rate to acute urethan dose and target cell number. *J. Nat. Cancer Inst. 69,* 851-547.

Thomas W.C. Jr., and O'Flaherty, E.J. (1982). The cardiotoxicity of carbon monoxide as a component of polymer pyrolysis smokes. *Toxicol. Appl. Pharmacol. 63,* 363-372.

O'Flaherty, E.J. and Thomas, W.C. (1982). The cardiotoxicity of hydrogen cyanide as a component of polymer pyrolysis smokes. *Toxicol. Appl. Pharmacol. 63,* 373-381.

O'Flaherty, E.J., Hammond, P.B., and Lerner, S.I. (1982). Dependence of apparent blood lead half-life on the length of previous lead exposure in humans. *Fund. Appl. Toxicol. 2,* 49-54.

O'Flaherty, E.J., Hammond, P.B. and Taylor, E. (1981). Renal reabsorption and secretion of δ-aminolevulinic acid in the rat. *Fund. Appl. Toxicol. 1,* 278-281.

Hammond, P.B., O'Flaherty, E.J. and Gartside, P.S. (1981). The impact of air lead on blood lead in man - a critique of the recent literature. *Food Cosmetics Toxicol. 19,* 631-638.

O'Flaherty, E.J., Hammond, P.B., Lerner, S.I. Hanenson, I.B. and Roda, S.M.B. (1980). The renal handling of δ-aminolevulinic acid in the rat in the human. *Toxicol. Appl. Pharmacol. 55,* 423-432.

Thomas W.C., Jr. and O'Flaherty, E.J. (1980). A system for exposing animals to smoke generated in a steady state fashion. *Environ. Res. 23,* 326-333.

Thomas, W.C., Jr., and O'Flaherty, E.J. (1979). Cytochrome c oxidase activity in tissues of rats exposed to polyurethane pyrolysis fumes. *Toxicol. Appl. Pharmacol. 49,* 463-472.

Manson, J.M. and O'Flaherty, E.J. (1978). Effects of cadmium on salamander survival and limb regeneration. *Environ. Res. 16,* 62-69.

O'Flaherty, E.J. and McCarty, C.P. (1978). Alterations of rat adipose tissue metabolism associated with dietary chromium supplementation. *J. Nutr. 108,* 321-328.

Petering, H.G., Murthy, L. and O'Flaherty, E.J. (1977). The influence of dietary co[redacted] zinc on rat lipid metabolism. *J. Agr. Food Chem. 25,* 1105-1109.

Abstracts and Presentations:

O'Flaherty, E.J. (1992). The toxicokinetics of lead. Presented to the Residue Technical Committee and Subcommittee on Trace Minerals in Foods, ILSI North America, Washington, DC, September 24, 1992.

O'Flaherty, E.J. (1992). Chromium: An essential and a toxic metal. ISTERH Third International Conference and NTES Fourth Nordic Conference on Trace Elements in Health and Disease, Stockholm (Huddinge), Sweden, May 25-29, 1992.

O'Flaherty, E.J. (1992). A calcium-based model of human lead kinetics. Presented at the NIH/U.S. EPA/Ross Laboratories conference, "Trace Element/Mineral Metabolism During Development," Washington, DC, June 8-10, 1992. Conference proceedings are to be published.

O'Flaherty, E.J. (1992). Physiologically-based pharmacokinetic models in developmental toxicology. Presented at the NRC/BEST/CRAM International Workshop on Statistical Methods in Developmental Toxicology, Ottawa, ON, May 21-22, 1992. Proceedings of the workshop to be published in *J. Risk Anal.*

O'Flaherty, E.J. (1992). A pharmacokinetic model for chromium. Presented at the Wright-Patterson Conference on Applications of Advances in Toxicology Risk Assessment, Wright-Patterson Air Force Base, Dayton, OH, May 19-21, 1992. Proceedings of the conference will be published in *Toxicol. Letters.*

O'Flaherty, E.J. (1992). Comparison of reference dose (RfD) with estimated safe and adequate daily dietary intake for chromium. Presented at the U.S. EPA/ATSDR/ILSI workshop, "Risk Assessment of Essential Elements," Herndon, VA, March 10-12, 1992. Workshop proceedings are to be published.

O'Flaherty, E.J. (1992). Physiologic changes during growth and development. Presented at the NAS/NRC workshop, "Pharmacokinetics: Defining Dosimetry for Risk Assessment," Washington, DC, March 4-6, 1992. Symposium proceedings to be published by NAS/NRC.

O'Flaherty, E.J. (1992). Application of a physiologically-based model to practical questions: Lead in adults. Presented at the U.S. EPA symposium, "Lead in Adults," Durham, NC, December 9-11, 1991. Symposium proceedings to be published in *Environ. Res.*

O'Flaherty, E.J. and Andriot, M.D. (1992). Predicting blood lead during human pregnancy. *The Toxicologist* 12, 212.

Himmelstein, M. W. and O'Flaherty, E.J. (1992). Physiologically-based toxicokinetic modeling of soluble uranium in the rat. *The Toxicologist* 12, 348.

O'Flaherty, E.J., Scott, W.J., Nau, H., and Beliles, R. (1992). Simulation of valproic acid kinetics in primate and rodent pregnancy by means of physiologically-based models. *Teratology* 45, 457.

Beliles, R., Scott, W., O'Flaherty, E.J. and Nau, H. (1992). Binding of methoxyacetic acid (MAA) in the mouse embryo. *Teratology* 45, 483.

O'Flaherty, E.J. (1992). Modeling bone mineral metabolism with special reference to calcium and lead. Presented at the workshop "Lead: Metabolism and Bone Deposition," Brookhaven National Laboratory, Brookhaven, NY, April 10-12, 1991. Workshop Proceedings to be published in *Neurotoxicology*.

O'Flaherty, E.J., Scott, W., and Beliles, R. (1991). A physiologically-based model of gestation in the monkey: framework for simulation of kinetics of reproductive toxicants. *Teratology* 43, 436.

O'Flaherty, E.J. (1991). Kinetics of lead disposition in humans. *The Toxicologist* 11, 52.

Himmelstein, M.W. and O'Flaherty, E.J. (1991). Dose-renal effect relationship during short-term exposure of rats to uranium (U). *The Toxicologist* 11, 236.

Hamilton, J.D. and O'Flaherty, E.J. (1991). Lead exposure and early endochondral bone growth. *The Toxicologist* 11, 82.

O'Flaherty, E.J. (1990). Physiologically based lead kinetics. Presented at the Twenty-Fourth Annual Conference on Trace Substances in Environmental Health, July, 1990.

O'Flaherty, E., Scott, W., Schreiner, C., and Beliles, R. (1990). A toxicokinetic model for pregnant rodents. Presented at the Annual Meeting of the Society of Teratology, June, 1990.

O'Flaherty, E.J. (1990). Development of a physiologically-based model for incorporation of lead into the rat fetus during gestation. *The Toxicologist* 10, 215.

Himmelstein, M.W. and O'Flaherty, E.J. (1990). Subcutaneous and intraperitoneal infusion of rats with hexavalent uranium: A pilot study. *The Toxicologist* 10, 157.

O'Flaherty, E.J. (1989). A physiologically-based toxicokinetic model for incorporation of lead into the growing skeleton. *The Toxicologist* 10, 56.

Hamilton, J. and O'Flaherty, E.J. (1989). Comparative effects of lead on growth in rats. *The Toxicologist* 9,100.

Himmelstein, M.W. and O'Flaherty, E.J. (1988). Modeling the effect of exposure duration on blood lead levels. *The Toxicologist* 8, 22.

Hamilton, J.D. and O'Flaherty, E.J. (1988). Lead exposure and skeletal develop-ment. *The Toxicologist* 8, 23.

O'Flaherty, E.J. (1988). A physiologically-based toxicokinetic model of the growing rat skeleton. *The Toxicologist* 8, 190.

Svirbely, J.E., Pesce, A.J., Singh, S., and O'Flaherty, E.J. (1988). Sulfamethoxazole/trimethoprim peritoneal barrier pharmacokinetics. *Clin. Chem.* 34, 1257.

Wadhwa, N.K., Schroeder, T.J., O'Flaherty, E., Pesce, A.J., Myre, S.A., Munda, R. and First, M.R. (1987). Interaction between erythromycin and cyclosporine in a kidney and pancreas allograft recipient. *Therapeutic Drug Monitoring* 9, 123-125 (short communication).

Wadhwa, N., Schroeder, T., O'Flaherty, E., Pesce, A. and First, M.R. (1986). Pharmacokinetics and drug interactions of cyclosporine (CYC) and erythromycin (ERY). *Clin. Res.* 34, 638A.

O'Flaherty, E.J. (1985). "Application of Pharmacokinetic Techniques to the Expression of Toxicant Dose," presented at a symposium on new issues in regulatory toxicology and health risk assessment at the Society of Toxicology Annual Meeting in San Diego, California, March 19-22.

Adams, W.D. and O'Flaherty, E.J. (1985). Urethane kinetics and pulmonary tumorigenesis. *The Toxicologist* 5, 39.

O'Flaherty, E.J. (1984). "The Rate of Decline of Blood Leads in the Lead Industry," presented at the Sixth Seminar for Physicians and Allied Health Professionals on Prevention of Occupational Lead Poisoning, sponsored and conducted by the Lead Industries Association, Inc., Palm Springs, California, October 10-12.

O'Flaherty, E.J. (1983). "Toxicokinetics and Dose," presented at a workshop on toxicokinetics in the safety evaluation of chemicals sponsored by the Electric Power Research Institute in San Diego, California, November 30 - December 2.

Sichak, S.P. and O'Flaherty, E.J. (1983). Urethane kinetics and pulmonary adenoma prevalence in Swiss-Cox mice. *The Toxicologist* 3, 145.

Sichak, S.P. and O'Flaherty, E.J. (1982). The kinetics of urethane metabolism in Swiss-Cox mice. *The Toxicologist* 2, 46.

Adams, W.D., O'Flaherty, E.J., and Taylor, E. (1982). Lead exposure and renal function in rats: is the rat a good model for lead induced nephropathy? *The Toxicologist* 2, 55.

Buben, J.A. and O'Flaherty, E.J. (1982). Relationship between trichloroethylene metabolism and its hepatotoxicity. *The Toxicologist* 2, 38.

Bonewitz, R.F., Foulkes, E.C., O'Flaherty, E.J. and Hertzberg, V.S. (1982). Kinetic evidence for glucocorticoid enhancement of jejunal zinc uptake. Intern. Symposium on Biological Aspects of Metals and Metal-related Diseases. Toronto, Ontario, October 19-22.

Hammond, P.B., Hong, C.D., O'Flaherty, E.J., Lerner, S.I. and Hanenson, I.B. (1981). The rat as an animal model of lead nephropathy. Presented at the Conference on Drugs and Environmental Toxicants, Pinehurst, North Carolina, March 18-20.

Buben, J.A. and O'Flaherty, E.J. (1981). Comparison of hepatotoxicities of trichloroethylene and perchloroethylene. Abstracts, Society of Toxicology Annual Conference, San Diego, California, March.

O'Flaherty, E.J. and Hammond, P.B. (1980). Renal reabsorption of δ-amino-levulinic acid in control and lead exposed rats. Abstracts, Society of Toxicology Annual Conference, Washington, DC, March.

Hammond, P.B., O'Flaherty, E.J. and Gartside, P.S. (1979). The impact of ambient air lead on uptake and retention of lead in man - a critique of the recent literature. Presented at the International Conference, Management and Control of Heavy Metals in the Environment. London, England, September, 1979, CEP Consultants Ltd., 26 Albany St., Edinburgh, UK, pp. 93-102.

Thomas, W.C., Jr., O'Flaherty, E.J., Bell, E.H. and Stemmer, K.L. (1978). The effect of polymer pyrolysis fumes on the activity of rat hepatic cytochrome c oxidase. Abstracts, Society of Toxicology Annual Conference, San Francisco, March 13-17.

Choudhury, H., Srivastava, L., Murthy, L., O'Flahety, E.J. and Petering, H. (1974). Effects of castration and adrenalectomy on serum zinc and copper. Proc. Vth Asia and Oceania Congress Endocrinol. 1, 216.

O'Flaherty, E.J., Murthy, L. and Petering, H.G. (1974). The influence of dietary manganese and chromium on serum ceruloplasmin activity, copper and zinc in male rats. *Fed. Proc.* 33, 668.

Murthy, L., O'Flaherty, E.J. and Petering, H.G. (1971). Effect of dietary levels of copper and zinc on serum lipids in rats. Summary Abstracts, 9th International Congress on Nutrition, Mexico City, p. 136.

Letters:

Hammond, P.B., O'Flaherty, E.J. and Gartside, P.S. (1982). Impact of air-lead on blood-lead in man. (Letter to the editor). *Food and Chemical Toxicology* 20, 493.

O'Flaherty, E.J. and Dourson, M.L. (1983). Cells of origin of lung tumors in mice. (Letter to the editor). *J. Natl. Cancer Inst.* 70, 991-992.

COMMENTS ON

"MADISON COUNTY LEAD EXPOSURE STUDY, GRANITE CITY, ILLINOIS"
by
R. Kimbrough, M. LeVois, and D. Webb

AND ON THE RELATED DOCUMENTS

"COMMENTS ON MADISON COUNTY LEAD EXPOSURE STUDY,
GRANITE CITY, ILLINOIS"
by
A.H. Marcus, K. Hogan, P. White, and P. Van Leeuwen

AND

"RESPONSE TO COMMENTS OF U.S. EPA REVIEWERS
REGARDING THE GRANITE CITY LEAD STUDY DRAFT REPORT"
by
M. LeVois

AND

"PRELIMINARY ASSESSMENT OF DATA FROM THE MADISON COUNTY
LEAD STUDY AND IMPLICATIONS FOR REMEDIATION
OF LEAD-CONTAMINATED SOIL"
by
A.H. Marcus

DOCUMENTS REVIEWED BY

Ellen J. O'Flaherty
November 22, 1994

THE STUDY

Madison County Lead Exposure Study, Granite City, Illinois (hereafter referred to as *Study*) is the report of a study of environmental lead exposure and blood lead concentrations in children, youths, and adults in Granite City, Illinois, carried out by the Illinois Department of Public Health (IDPH) in late 1991. The primary hypothesis tested was whether lead in soil contributed significantly to blood lead levels in children less than 6 years old. Participants were recruited from four residential areas comprising rough concentric rings around a closed secondary lead smelter, the NL/Taracorp site. Preliminary monitoring data had indicated that soil lead concentrations decreased with distance from the smelter. The innermost area is a Superfund site. Because no suitable control residential area (e.g., similar housing stock and similar socioeconomic status of residents) could be identified, the four residential sampling areas served only to achieve a fairly representative range of soil lead values. Measured soil lead values, not sampling areas, were used in the statistical analysis.

Blood lead concentrations and the content of lead in environmental samples of dust, paint, soil, and drinking water were the key measured lead variables. Other variables, such as indicators of socioeconomic status (SES), condition of the exterior of the house, and condition of the interior paint were elicited by observation or by questionnaire.

Data analysis proceeded through a series of logical steps. Bivariate analyses were applied initially to the data from groups of children with blood lead concentrations greater than/equal to or less than 10 ug/dl and from groups of children living in regions with composite soil lead levels greater than/equal to or less than 500 ppm. The purpose of the bivariate analyses was to provide a simple first screening to eliminate variables not associated with blood lead. This preliminary data evaluation was followed by multiple regression analyses whose purposes were (1) to help identify variables that had utility in predicting blood lead concentrations, (2) to identify by means of maximum regression coefficient (R^2) improvement analysis the subset of variables with the greatest predictive utility, and (3) by means of hierarchical regression modeling, to evaluate the contribution of soil lead to blood lead and house dust lead.

The preliminary bivariate data analysis narrowed the list of potential predictor variables to lead in indoor paint, soil lead, dust lead and dust loading, distance from the NL/Taracorp site, parents' level of education, parents' income, variables related to smoking, number of hours spent outdoors by the child per day, number of baths taken per week, and the categorical variables presence/absence of air conditioning, renting/owning, condition of exterior of residence, and refinishing of the residence within the past year or of furniture within the past three months.

Stepwise regression analysis was used to select and assign level of importance to the critical predictors. The two primary predictors were found to be dust lead and distance from the smelter site, the two together accounting for 21% of the total variance in blood lead. Other predictors were the parents' level of education, number of cigarettes smoked per day, rent/own home, refinishing activities, ethnicity, dust load, age, drinking water lead concentration, distance, and number of hours of outdoor play per day. Together with dust lead and distance from the smelter site, these variables accounted for 35% of the blood lead

variance.

Hierarchical regression focused specifically on the contribution of paint and soil lead to blood lead, using a very small set of predictor variables so as not to overadjust by confounding. Water lead level, house paint lead levels, recent refinishing activities, and condition of the residence were the only potentially confounding variables introduced. House dust was not included as a potential confounder, since it was a pathway for the two primary lead sources, soil and paint. Soil lead alone was found to account for 3% of the variance in blood lead, while the condition of the house and the amount of lead in paint together accounted for about 11% of the variance in blood lead. Soil lead accounted for 6% of the dust lead variance, while indoor and outdoor paint lead levels and the condition of the residence together accounted for 26% of the variance in dust lead.

The conclusion is that at this site, soil was less important as an ultimate lead source than interior and exterior paint. Both soil lead and paint lead were found to contribute to dust lead, a pathway of exposure. Building condition was an important modifier of the contribution of paint lead to dust lead. Paint lead levels and building condition together accounted for about four times as much (26%) of the variance in dust lead as soil lead did (6%).

THE COMMENTS AND THE RESPONSE

Comments on Madison County Lead Exposure Study, Granite City, Illinois (hereafter referred to as *Comments*) is a detailed analysis of the study report. In general, many of the points raised are conventional, almost textbook recommendations for the conduct of environmental studies; however, often they miss the mark. Sometimes they do not seem to be connected with the section of *Study* being commented upon at all. The fundamental problem is that the reviewers do not seem to recognize that the study design used here was not the conventional environmental epidemiology study design with an exposed community group and a control group. In addition, a number of the points they raise had already been raised and discussed by the authors of the study report, suggesting that the reviewers did not read the report in full.

I have divided my comments on *Comments* in accordance with the structure adopted in both *Study* and *Comments*. I also refer to *Response to Comments of U.S. EPA Reviewers Regarding the Granite City Lead Study Draft Report* (hereafter referred to as *Response*).

1. IMPLEMENTATION OF STUDY DESIGN

1.1 Recruitment of Subjects

This section contains some general comments about avoidance of bias in recruiting. The stated concern that the participation rate might have been lower in areas with higher exposure and lower resident socioeconomic status is not supported by the observation that the participation rate was about equal in three of the four areas, but lower in the area with the

lowest lead exposure — a point already made and discussed in *Study*.

1.2 Omission of Pontoon Beach Subjects

Nothing would have been gained by including the small group of Pontoon Beach subjects, whose housing was entirely different from the housing in Granite City. A control group was not needed for the statistical analysis used.

1.3 Resampling of Children with Elevated Blood Lead

The logic of the first paragraph is not clear. How does bias due to inclusion of siblings follow from the "regression to the mean" problem? And what does the inclusion of some sibling measurements have to do with the conclusion that was reached by the authors, which was simply that counseling resulted in a drop in blood lead in those children whose families were counseled? Those drops were almost 50%. *Comments* notes that in other studies, children tested in winter have had lower blood lead concentrations, typically by about 30%, than children who were tested at the summertime peak, and suggests that this phenomenon invalidates the observation that counseling was followed by reductions in blood lead. In fact, *Study* discusses the seasonal fluctuation in blood lead concentrations in children in some detail, and points out that the first blood samples were taken when the time of the summer peak was already past.

The authors of *Study* made no claim that they were testing a hypothesis about the effectiveness of counseling and education in reducing blood lead. It is unnecessary for the reviewers to outline a study that could be used to test such a hypothesis, and irresponsible for them to suggest that "...the resampled children in the Madison County study are used to reach some very broad and general conclusions....". The authors have not generalized from the data from the resampled children. They have simply, and correctly, stated an observation.

2. FIELD SAMPLING AND ANALYSIS OF SAMPLES

2.1-2.3 I have no comments on these sections.

2.4 House Condition and Paint Condition

I agree with the reviewers that clearer statements of the criteria for classification of housing condition as good, fair, or poor would have been helpful. In addition, it is not always immediately clear in *Study* whether exterior or interior paint condition, or both, are included in a variable to be tested. These are minor points related to clarity of presentation, unrelated to study design or conduct.

3.STATISTICAL ANALYSIS OF DATA

3.1 Dependence on Age

It is pointed out in *Response* that blood lead models were not adjusted for age, as the reviewers mistakenly thought.

3.2 Inadequate Spatial Resolution of Demographics and Lead Exposure

The reviewers seem to have missed the point entirely here. They are still viewing the study as one in which different groups are to be compared: groups that should be identical in all respects except magnitude of lead exposure. They take issue with use of concentric geographic rings to define subject groups, but in fact subject groups were not used in the study. They remark that older housing may contain a disproportionate number of families with more than one child less than 6 years old, when it was clearly stated in the report that the study unit was the child, not the household, for most of the statistical analyses; and was also shown that outcome was not affected by using either all children in each family, or only the child with the highest or lowest blood lead concentration (*Study*, Table 13 and p. 44). The reviewers then proceed to suggest an alternate study design, one that might distinguish among sub-regions with different exposure characteristics. The purpose of this exercise is not entirely clear, but it may have been to demonstrate that the percentage of children with blood lead concentrations above 10ug/dl is not uniform throughout the study area, an observation that is interesting, but irrelevant in view of the study design. Why this is considered by the reviewers to be "absolutely vital information," as stated in the last sentence of this section, is also not clear.

The reviewers also point out in connection with this observation that "...distance alone does not describe the distribution of elevated blood lead in the study area." The authors have not claimed that it does, and again this point is irrelevant in view of the study design.

3.3 The Regression Model

Response deals quite adequately with this silly misinterpretation.

3.4 Contribution of Soil Lead to Blood Lead and to Dust Lead

Again, the authors of *Study* have explained very clearly in *Response* the failure of the reviewers in *Comments* to understand the purpose and process of hierarchical regression.

3.5 Multi-Media and Multi-Pathway Lead Exposure

Path analysis would have been one technique for dealing with these data, a technique that the authors did not choose to use. But why do the reviewers believe that, "This approach would be far more useful in identifying appropriate goals for environmental intervention."? As pointed out in *Response*, there is again evidence in this section of *Comments* that the reviewers have failed to understand hierarchical regression analysis and its

interpretation.

3.6 Individual Behavioral Variables

Response addresses this point particularly well, and sums up what I believe is the central problem in *Comments*: "...adding behavioral or other variables to a hierarchical regression model can only reduce the variance accounted for by soil. The reviewer seems to want to find some set of variables that lead to a higher simultaneous parameter estimate for soil, regardless of how little variance is explained by the individual variables, or how all of the other environmental variables are affected by the factors the reviewer wants included in a single analysis."

3.8 Multicolinearity

Another comment effectively rebutted in *Response*. It is pointed out that this comment is antithetical to the previous one!

3.9 Biases Due to Predictor Measurement

Again, a "textbook" comment that is not uniquely relevant to this study. It almost seems that the authors of *Comment* are attempting to provide a review of design difficulties and statistical uncertainties inherent to all epidemiological studies, and to associate these uncertainties with this particular study whether or not they are related to the study design and to the use of hierarchical regression analysis techniques.

4. PRESENTATION OF RESULTS

4.1 Statistical Tables

No comments.

4.2 Graphs

The graphs in Figures 2a and 2b are perfectly clear. They are not histograms.

4.3 Maps

No comments.

4.4 Confidence Intervals

This comment is adequately rebutted in *Response*.

5. INTERPRETATION AND CONCLUSIONS

1. This is a matter of interpretation.

2. Child age was *not* entered as a monotone predictor of blood lead. This criticism is inappropriate.

3. While clusters of cases representing elevated lead exposure were laboriously extracted by the reviewers from the map included in *Study*, it is not clear what the function of this exercise was. The negative association of distance with blood lead concentration was documented and reported in *Study* (pp. 38, 49).

4. The reviewers miss the point again. The *pattern* of soil lead distribution was not relevant to the study, since *actual* soil lead levels rather than radial distances were used in the analysis.

5. This comment is wrong. It is effectively rebutted in *Response*.

6. Again, this comment is effectively rebutted in *Response*.

7. The comment in *Response* is appropriate.

8. Again, the comment in *Response* is appropriate.

9. This criticism is wrong. Its gratuitous suggestion that the data analysis should be redone would be suspect if only because of the associated and wholly unsupported statement that, "The reported results are highly compatible with the causal model we proposed in Section 3.4, that lead in soil is an important indirect source of lead in blood through the soil-to-dust pathway."

THE REASSESSMENT

The report, *Preliminary Assessment of Data from the Madison County Lead Study and Implications for Remediation of Lead-Contaminated Soil*, by Allan Marcus (hereafter called *Reassessment*), is difficult to characterize. Dr. Marcus states that this report is very preliminary and that a more detailed report will be prepared that will describe the methods used in the analyses and will present a complete set of results together with the basis for his conclusions. Nonetheless, there are fundamental flaws in the analysis as it is presented here, along with the large gaps that are to be filled at a later date.

Section 1. Here, Dr. Marcus states the goals of this data reanalysis. Two of them, to provide site-specific information about relevant parameters and to evaluate the proposed soil remediation level "using this recent information," are not met, as will be detailed below.

Section 2. In Section 2, Dr. Marcus reanalyzes the data from the IDPH study. The "reanalysis" consists, first, of restatements of the fractions of the study group of children with blood lead concentrations above specified cutoff levels, starting with 10 ug/dl. These fractions are given in *Study*. It is not clear what Dr. Marcus intends to imply by repeatedly underscoring "in the study" when he refers to households or children with particular characteristics (points 2, 7, and 8). These restatements are followed by a series of graphs in which the logarithms of a number of variables such as mean blood lead concentration and environmental lead concentrations are plotted against distance from the NL/Taracorp site within 10 concentric rings around the site, each about 1/8 mile in thickness. On the basis that blood lead, soil lead, and dust lead all show similar patterns of decreasing concentration with increasing distance from the NL/Taracorp site, he concludes that both soil lead and dust lead are contributors to blood lead (point 3). However reasonable it may seem to be on the surface, such a conclusion is absolutely unjustifiable. Simple correlations cannot support such a conclusion. Following the detailed criticism in *Comments* of every aspect of the statistical methods used in *Study*, Dr. Marcus' leap of faith here is particularly egregious.

Most of the remainder of his conclusions in this section are unexceptionable, although most of them are basically restatements of the data, some of them seem to be general statements with no necessary relationship to the present data set, and some of them verge perilously close to personal opinion unsupported by data from the study ("Households in the study with the most children *and the fewest resources to cope with lead poisoning* are located closest to the NL site." (italics added)). The purpose of one other statement is unclear, however: point 9. Dr. Marcus points out that the participation rate was lower in the zone farthest from the NL/Taracorp site, and expresses concern that the sample of children may therefore not be representative of the community. In view of the fact that the four zones were not all the same size, it is not immediately clear whether this concern is justified. However, more to the point is that such a maldistribution would, if it had any effect at all, have skewed the distribution of blood lead concentrations in the direction of higher rather than lower values.

On the basis, apparently, that the linear regression relationships that were so strongly criticized in *Comments* are statistically significant and that the data set is similar to those from other EPA sites, Dr. Marcus judges that these data can be used for evaluating childhood lead exposure in Madison County.

Most troubling about this entire "reanalysis," apart from its cavalier mixing of correlations, general statements, and personal judgment, all unleavened with any statistics (although presumably these will be made available later), is that it is based entirely on the distance of concentric rings from the plant site. In *Comments*, Section 3.2, with Dr. Marcus as the first author, it is bluntly stated that, "The division of the study area into concentric rings is not defensible." Surely, what was not defensible then should not be defensible now. Unfortunately, one is left with the rather strong impression that features of the IDPH study that were judged unacceptable when they seemed to point to one conclusion have become acceptable when they are used to buttress a different conclusion.

Section 3. This analysis is little more than a running of the IEUBK model with its default parameters. Site-specific exposure data were not used. One site-specific parameter

was examined in a sensitivity analysis. The statement that, "Site-specific parameters were based on our judgment and analyses that the NL site had many points of similarity to the calibration site, Midvale,...." is disingenuous in the extreme. Midvale is a town, not an urban community, associated with a mining site, not a smelter. That the default parameters of the IEUBK model give a blood lead concentration distribution that is similar (although there is considerable discrepancy in the lower tail) to the observed distribution in the Granite City study does not imply that another set of parameters, perhaps one that was truly site-specific, would not give an equally good or even better match.

A small sensitivity analysis was carried out, varying the value of the soil-to-dust coefficient using the default value and three additional values based on "statistical analyses from study data." It is not stated what kind of statistical analysis was used. The default value of the soil-to-dust coefficient "...was judged to be appropriate, and also provided a very good fit to the child blood lead data...." In addition, the two larger values of the soil-to-dust coefficient tested, including the default value, "...are more appropriate for risk assessment, more realistic for properties of the site, and provide a good fit to the data." Since two of the three values stated to have been generated from study data were excluded by this statement, it is hard to see how the larger values are more representative of properties of the site. Curiously, however, the final suggested range of soil cleanup values *includes* the values generated by use of the two previously-excluded lower soil-to-dust coefficients, but *excludes* the values generated by use of the previously-recommended larger soil-to-dust coefficients. We are not told whether the two lower soil-to-dust coefficients also gave good fits to the distribution of children's blood lead concentrations.

The overall impression given by this entire "reanalysis" is that the recommended soil remediation level was predetermined. The "reanalysis" is superficial and careless, and bears little if any relationship to the data from the IDPH study.

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Place of Birth: Cincinnati, Ohio

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Education:
Ph.D., Chemistry, Yale University, 1964
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EXPERIENCE:

1984-:	Associate Professor of Environmental Health, University of Cincinnati College of Medicine. Director, Toxicology Training Program, 1987-present.
1982-1984:	Research Associate Professor of Environmental Health, University of Cincinnati College of Medicine.
1975-1982:	Assistant Professor of Environmental Health, University of Cincinnati College of Medicine.
1970-75 and 1966-68:	Senior Research Associate in Environmental Health, University of Cincinnati College of Medicine.
1968-70:	On leave of absence.

HONORS:

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PROFESSIONAL SOCIETIES AND RELATED ACTIVITIES:

Society of Toxicology. Member of Editorial Board of the official journal of the Society, "Fundamental and Applied Toxicology," 1983-1985.

Associate Editor of the other official journal, "Toxicology and Applied Pharmacology," 1985-1990. Councillor of the Ohio Valley Section of the Society, 1987-present.

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Curriculum Vitae
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Member, College of Medicine Grievance Committee, 1985-1987

Secretary, Faculty Forum, 1982-83.

Departmental representative to Faculty Forum Executive Committee, 1983-present.

Departmental representative to College Committee on Graduate Education, 1990-present.

College of Medicine representative to Faculty Senate, 1987-1989.

Member, Degrees Committee of the Department of Environmental Health, University of Cincinnati, 1975-1987; chair, 1985-1987.

Member, Library Committee of the Department of Environmental Health, 1980-1985.

Member, Art Committee of the College of Medicine, 1979-1983.

Member, Commencement Committee of the University of Cincinnati, 1974-77.

RELEVANT SCIENTIFIC ADVISORY AND CONSULTING EXPERIENCE:

Member, Ohio EPA Surface Water Task Force, 1987-present.

Participant in Cincinnati EPA - Department of Environmental Health Cooperative Agreement for criteria document preparation and review, 1987-present.

Author of pharmacokinetic, risk assessment, and criterion formulation sections of a number of EPA water quality criteria documents including Iron and Its Compounds (1981), Dibenzofurans (1981), Endrin (1982), Toxaphene (1983), and the multimedia document Aluminum (1989).

Reviewer or member of external review panel for a variety of other EPA water quality criteria documents including Ammonia (1982), Ethylbenzene (1985), 2,4,5-Trichlorophenoxy propionic acid (1985), and the multimedia document Polycyclic Aromatic Hydrocarbons (1987).

Author of review of 2-ethylhexanol toxicity and contributor to an evaluation of literature on human exposure to 2,4-dichlorophenoxyacetic acid for the 2,4-D industries association, 1986-1987.

Reviewer for water quality criteria document, Perchloroethylene, for California State EPA, 1987.

Consultant to the Lead Industries Association on an ad hoc basis, primarily concerning the kinetics of changes in blood lead concentrations during OSHA-mandated removal of lead workers from workplace situations leading to unacceptable blood lead concentrations.

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GRADUATE AND OTHER TEACHING EXPERIENCE:

Kinetics for the Health Sciences - A 3-graduate-credit course covering linear and nonlinear kinetics in single- and multi-compartment systems; acute and chronic exposure; dose-effect and dose-response relationships; physiologically-based pharmacokinetic models; and application of these principles to problems in research and in risk assessment.

Lectures, generally covering kinetics and risk assessment applications, in other graduate courses such as Risk Assessment, Survey of Toxicology, and Fundamentals of Environmental Toxicology.

Course Director for two courses in Continuing Education Program of Department: Risk Assessment: Principles and Applications, and Advanced Risk Assessment: Biological and Environmental Modeling. Lectures in other Continuing Education courses such as Biological Monitoring and the Mini-residency course for physicians.

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Committees:

Qualifying examination committees for Dana Laurie, Michael Dourson, William Thomas, Stephen Henne, Richard Costlow, Barry Blakley, John Buben, Gary Thompson (Pharmacy), Rosalind Schoof, Ray York, Dan Talsma, Paul Sichak, Wayne Adams, Orisa Igwe, Loretta Schuman, James Sherman, John Hamilton, Matthew Himmelstein, Patricia Plews, Candace Lippoli, Michelle Andriot.

Dissertation committees for Jon Reid, Michael Dourson, William Thomas, Paul Ringhand (Pharmacy), Gregory Hammer (Pharmacy), Timothy Hardt (Pharmacy), Alan Gerstein (Microbiology), Carol Silberstein, John Buben, Paul Sichak, Orisa Igwe, Mark Toraason, Laureano Leon, Jerry Ann Ward, Joseph Svirbely, Helen Goeden, Deborah Oudiz, Wayne Adams, William Cibulas, Christopher Bevan, Jonathan Meier, John Hamilton, Matthew Himmelstein, Richard Cassidy, Michelle Andriot.

Curriculum Vitae
Ellen J. O'Flaherty

RESEARCH INTERESTS:

Application of established kinetic principles to new areas; e.g., toxicology rather than pharmacology. Current work: Kinetics of lead transfer among body compartments and its significance relative to lead toxicity. Development and testing of new kinetic approaches to biological questions.

PUBLICATIONS AND PRESENTATIONS:

Books:

O'Flaherty, E.J. (1981). *Toxicants and Drugs: Kinetics and Dynamics*. John Wiley and Sons, Inc.

Book Chapters:

O'Flaherty, E.J. (1987). Modeling: An Introduction. In: *Pharmacokinetics in Risk Assessment, Drinking Water and Health*, Volume 8: pp. 27-35. National Research Council, National Academy Press, Washington, D.C.

O'Flaherty, E.J. (1986). Toxicokinetics and Dose. In: *Proceedings: New Directions on the Extrapolation of Health Risks from Animals to Man*, Volume 2: Toxicokinetics", Chapter 5, pp 5-1 - 5-10. Electric Power Research Institute, Palo Alto, CA 94304. Proceedings of a workshop held November 30-December 2, 1983, San Diego, CA.

O'Flaherty, E.J. (1985). Differences in metabolism at different dose levels. Chapter 3, In: *Toxicological Risk Assessment*, Volume I: Biological and Statistical Criteria, ed. D.B. Clayson, D.R. Krewski, and I.C. Munro, CRC Press, pp. 53-90.

O'Flaherty, E.J. (1985). General principles of toxicology. Chapter 3, In: *Industrial Toxicology - Safety and Health Applications in the Workplace*. Belmont, California, Lifetime Learning Publications, pp. 27-57.

O'Flaherty, E.J. (1984). *Pharmacokinetics*. In: *Risk Assessment and Risk Assessment Methods: The State-of-the-Art*. Division of Policy Research and Analysis, National Science Foundation, Washington, D.C. 20550, pp. 283-320.

Hammond, P.B., Hong, C.D., O'Flaherty, E.J., Lerner, S.I. and Hanenson, I.B. (1982). *The Rat as an Animal Model of Lead Nephropathy*. In: *Nephrotoxic Mechanisms of Drugs and Environmental Toxins*. George Porter, editor, Plenum Publishing Corporation, New York, pp. 267-277.

Bonewitz, R.F., Foulkes, E.C., O'Flaherty, E.J. and Hertzberg, V. (1982). *The Effect of Dexamethasone on the Kinetics of Jejunal Zinc Uptake and Metallo-thionein Synthesis in the Rat*. In: *Biological Roles of Metallothionein*, E. C. Foulkes, editor, Elsevier/North Holland, Inc., New York, pp. 203-214.

Curriculum Vitae
Ellen J. O'Flaherty

Papers in Refereed Journals:

O'Flaherty, E.J. (1992). Physiologically-based models for bone-seeking elements. IV. Kinetics of lead disposition in humans. In press, *Toxicol. Appl. Pharmacol.*

O'Flaherty, E.J., Scott, W., Schreiner, C. and Beliles, R.P. A physiologically-based kinetic model of rat and mouse gestation: disposition of a weak acid. *Toxicol. Appl. Pharmacol.*, 112:245-256, 1992.

O'Flaherty, E.J. (1991). Physiologically-based models for bone-seeking elements. I. Rat skeletal and bone growth. *Toxicol. Appl. Pharmacol.*, 111:299-312.

O'Flaherty, E.J. (1991). Physiologically-based models for bone-seeking elements. II. Kinetics of lead disposition in rats. *Toxicol. Appl. Pharmacol.*, 111:313-331.

O'Flaherty, E.J. (1991). Physiologically-based models for bone-seeking elements. III. Human skeletal and bone growth. *Toxicol. Appl. Pharmacol.*, 111:332-341.

O'Flaherty, E.J. (1991). Physiologically based lead kinetics. *Trace Subst. Environ. Health 24*, 44-54.

O'Flaherty, E.J. (1989). Interspecies conversion of kinetically equivalent doses. *Risk Analysis 9*, 587-598.

O'Flaherty, E.J. (1989). Application of pharmacokinetic principles to exposure to chemical mixtures. *Toxicol. Industr. Health 5*, 667-680.

Svirbely, J.E., Singh, S., Pesce, A.J., and O'Flaherty, E.J. (1988). Sulfa-methoxazole and trimethoprim peritoneal barrier transfer pharmacokinetics. *Clinical Pharmacokinetics 16*, 317-325.

O'Flaherty, E.J. (1988). A physiologically based model of skeletal growth in the rat. *Toxicol. Letters 43*, 85-95.

York, R.G., O'Flaherty, E., Scott, W.J. and Shukla, R. (1987). Alteration of effective exposure of dam and embryo to caffeine and its metabolites by treatment with beta naphthoflavone. *Appl. Pharmacol. 88*, 282-293

O'Flaherty, E.J., Adams, W.D., Hammond, P.B. and Taylor, E. (1986). Resistance of the rat to development of lead-induced renal functional deficits. *J. Toxicol. Environ. Hlth. 18*, 61-75.

O'Flaherty, E.J. (1986). The rate of decline of blood lead in lead industry workers during medical removal: The effect of job tenure. *Fund. Appl. Toxicol. 6*, 372-380.

Buben, J.A. and O'Flaherty, E.J. (1985). Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: A dose-effect study. *Toxicol. Appl. Pharmacol. 78*, 105-122.

Sichak, S.P. and O'Flaherty, E.J. (1984). Consideration of the mechanism of pulmonary adenogenesis in urethane-treated Swiss mice. *Toxicol. Appl. Pharmacol. 76*, 397-402.

Curriculum Vitae
Ellen J. O'Flaherty

O'Flaherty, E.J. and Sichak, S.P. (1983). The kinetics of urethane elimination in the mouse. *Toxicol. Appl. Pharmacol. 68,* 354-358.

Hirsch, K.S., Wilson, J.G., Scott, W.J. O'Flaherty, E.J. (1983). Acetazolamide teratology and its association with carbonic anhydrase inhibition in the mouse. *Teratogenesis, Carcinogenesis, and Mutagenesis, 3,* 133-144.

Bonewitz, R.F., Foulkes, E.C., O'Flaherty, E.J. and Hertzberg, V.S. (1983). Kinetics of Zn absorption by rat jejunum: Effects of adrenalectomy and dexa-methasone. *Amer. J. Physiology 244,* G314-320.

O'Flaherty, E.J. and Dourson, M.L. (1982). Relationship between urethane dose rate and adenoma latency: relevance of tumor growth rate and target cell number. *J. Natl. Cancer Inst. 69,* 859-865.

Dourson, M.L. and O'Flaherty, E.J. (1982). Relationship of lung adenoma prevalence and growth rate to acute urethan dose and target cell number. *J. Nat. Cancer Inst. 69,* 851-547.

Thomas W.C. Jr., and O'Flaherty, E.J. (1982). The cardiotoxicity of carbon monoxide as a component of polymer pyrolysis smokes. *Toxicol. Appl. Pharmacol. 63,* 363-372.

O'Flaherty, E.J. and Thomas, W.C. (1982). The cardiotoxicity of hydrogen cyanide as a component of polymer pyrolysis smokes. *Toxicol. Appl. Pharmacol. 63,* 373-381.

O'Flaherty, E.J., Hammond, P.B., and Lerner, S.I. (1982). Dependence of apparent blood lead half-life on the length of previous lead exposure in humans. *Fund. Appl. Toxicol. 2,* 49-54.

O'Flaherty, E.J., Hammond, P.B. and Taylor, E. (1981). Renal reabsorption and secretion of δ-aminolevulinic acid in the rat. *Fund. Appl. Toxicol. 1,* 278-281.

Hammond, P.B., O'Flaherty, E.J. and Gartside, P.S. (1981). The impact of air lead on blood lead in man - a critique of the recent literature. *Food Cosmetics Toxicol. 19,* 631-638.

O'Flaherty, E.J., Hammond, P.B., Lerner, S.I. Hanenson, I.B. and Roda, S.M.B. (1980). The renal handling of δ-aminolevulinic acid in the rat in the human. *Toxicol. Appl. Pharmacol. 55,* 423-432.

Thomas W.C., Jr. and O'Flaherty, E.J. (1980). A system for exposing animals to smoke generated in a steady state fashion. *Environ. Res. 23,* 326-333.

Thomas, W.C., Jr., and O'Flaherty, E.J. (1979). Cytochrome c oxidase activity in tissues of rats exposed to polyurethane pyrolysis fumes. *Toxicol. Appl. Pharmacol. 49,* 463-472.

Manson, J.M. and O'Flaherty, E.J. (1978). Effects of cadmium on salamander survival and limb regeneration. *Environ. Res. 16,* 62-69.

O'Flaherty, E.J. and McCarty, C.P. (1978). Alterations of rat adipose tissue metabolism associated with dietary chromium supplementation. *J. Nutr. 108,* 321-328.

Petering, H.G., Murthy, L. and O'Flaherty, E.J. (1977). The influence of dietary copper and zinc on rat lipid metabolism. *J. Agr. Food Chem. 25,* 1105-1109.

Abstracts and Presentations:

O'Flaherty, E.J. (1992). The toxicokinetics of lead. Presented to the Residue Technical Committee and Subcommittee on Trace Minerals in Foods, ILSI North America, Washington, DC, September 24, 1992.

O'Flaherty, E.J. (1992). Chromium: An essential and a toxic metal. ISTERH Third International Conference and NTES Fourth Nordic Conference on Trace Elements in Health and Disease, Stockholm (Huddinge), Sweden, May 25-29, 1992.

O'Flaherty, E.J. (1992). A calcium-based model of human lead kinetics. Presented at the NIH/U.S. EPA/Ross Laboratories conference, "Trace Element/Mineral Metabolism During Development," Washington, DC, June 8-10, 1992. Conference proceedings are to be published.

O'Flaherty, E.J. (1992). Physiologically-based pharmacokinetic models in developmental toxicology. Presented at the NRC/BEST/CRAM International Workshop on Statistical Methods in Developmental Toxicology, Ottawa, ON, May 21-22, 1992. Proceedings of the workshop to be published in *J. Risk Anal.*

O'Flaherty, E.J. (1992). A pharmacokinetic model for chromium. Presented at the Wright-Patterson Conference on Applications of Advances in Toxicology Risk Assessment, Wright-Patterson Air Force Base, Dayton, OH, May 19-21, 1992. Proceedings of the conference will be published in *Toxicol. Letters.*

O'Flaherty, E.J. (1992). Comparison of reference dose (RfD) with estimated safe and adequate daily dietary intake for chromium. Presented at the U.S. EPA/ATSDR/ILSI workshop, "Risk Assessment of Essential Elements," Herndon, VA, March 10-12, 1992. Workshop proceedings are to be published.

O'Flaherty, E.J. (1992). Physiologic changes during growth and development. Presented at the NAS/NRC workshop, "Pharmacokinetics: Defining Dosimetry for Risk Assessment," Washington, DC, March 4-6, 1992. Symposium proceedings to be published by NAS/NRC.

O'Flaherty, E.J. (1992). Application of a physiologically-based model to practical questions: Lead in adults. Presented at the U.S. EPA symposium, "Lead in Adults," Durham, NC, December 9-11, 1991. Symposium proceedings to be published in *Environ. Res.*

O'Flaherty, E.J. and Andriot, M.D. (1992). Predicting blood lead during human pregnancy. *The Toxicologist* 12, 212.

Himmelstein, M. W. and O'Flaherty, E.J. (1992). Physiologically-based toxicokinetic modeling of soluble uranium in the rat. *The Toxicologist* 12, 348.

O'Flaherty, E.J., Scott, W.J., Nau, H., and Beliles, R. (1992). Simulation of valproic acid kinetics in primate and rodent pregnancy by means of physiologically-based models. *Teratology* 45, 457.

Beliles, R., Scott, W., O'Flaherty, E.J. and Nau, H. (1992). Binding of methoxyacetic acid (MAA) in the mouse embryo. *Teratology* 45, 483.

O'Flaherty, E.J. (1992). Modeling bone mineral metabolism with special reference to calcium and lead. Presented at the workshop "Lead: Metabolism and Bone Deposition," Brookhaven National Laboratory, Brookhaven, NY, April 10-12, 1991. Workshop Proceedings to be published in *Neurotoxicology*.

O'Flaherty, E.J., Scott, W., and Beliles, R. (1991). A physiologically-based model of gestation in the monkey: framework for simulation of kinetics of reproductive toxicants. *Teratology* 43, 436.

O'Flaherty, E.J. (1991). Kinetics of lead disposition in humans. *The Toxicologist* 11, 52.

Himmelstein, M.W. and O'Flaherty, E.J. (1991). Dose-renal effect relationship during short-term exposure of rats to uranium (U). *The Toxicologist* 11, 236.

Hamilton, J.D. and O'Flaherty, E.J. (1991). Lead exposure and early endochondral bone growth. *The Toxicologist* 11, 82.

O'Flaherty, E.J. (1990). Physiologically based lead kinetics. Presented at the Twenty-Fourth Annual Conference on Trace Substances in Environmental Health, July, 1990.

O'Flaherty, E., Scott, W., Schreiner, C., and Beliles, R. (1990). A toxicokinetic model for pregnant rodents. Presented at the Annual Meeting of the Society of Teratology, June, 1990.

O'Flaherty, E.J. (1990). Development of a physiologically-based model for incorporation of lead into the rat fetus during gestation. *The Toxicologist* 10, 215.

Himmelstein, M.W. and O'Flaherty, E.J. (1990). Subcutaneous and intraperitoneal infusion of rats with hexavalent uranium: A pilot study. *The Toxicologist* 10, 157.

O'Flaherty, E.J. (1989). A physiologically-based toxicokinetic model for incorporation of lead into the growing skeleton. *The Toxicologist* 10, 56.

Hamilton, J. and O'Flaherty, E.J. (1989). Comparative effects of lead on growth in rats. *The Toxicologist* 9,100.

Himmelstein, M.W. and O'Flaherty, E.J. (1988). Modeling the effect of exposure duration on blood lead levels. *The Toxicologist* 8, 22.

Hamilton, J.D. and O'Flaherty, E.J. (1988). Lead exposure and skeletal develop-ment. *The Toxicologist* 8, 23.

O'Flaherty, E.J. (1988). A physiologically-based toxicokinetic model of the growing rat skeleton. *The Toxicologist* 8, 190.

Svirbely, J.E., Pesce, A.J., Singh, S., and O'Flaherty, E.J. (1988). Sulfamethoxazole/trimethoprim peritoneal barrier pharmacokinetics. *Clin. Chem.* 34, 1257.

Wadhwa, N.K., Schroeder, T.J., O'Flaherty, E., Pesce, A.J., Myre, S.A., Munda, R. and First, M.R. (1987). Interaction between erythromycin and cyclosporine in a kidney and pancreas allograft recipient. *Therapeutic Drug Monitoring* 9, 123-125 (short communication).

Wadhwa, N., Schroeder, T., O'Flaherty, E., Pesce, A. and First, M.R. (1986). Pharmacokinetics and drug interactions of cyclosporine (CYC) and erythromycin (ERY). *Clin. Res.* 34, 638A.

O'Flaherty, E.J. (1985). "Application of Pharmacokinetic Techniques to the Expression of Toxicant Dose," presented at a symposium on new issues in regulatory toxicology and health risk assessment at the Society of Toxicology Annual Meeting in San Diego, California, March 19-22.

Adams, W.D. and O'Flaherty, E.J. (1985). Urethane kinetics and pulmonary tumorigenesis. *The Toxicologist* 5, 39.

O'Flaherty, E.J. (1984). "The Rate of Decline of Blood Leads in the Lead Industry," presented at the Sixth Seminar for Physicians and Allied Health Professionals on Prevention of Occupational Lead Poisoning, sponsored and conducted by the Lead Industries Association, Inc., Palm Springs, California, October 10-12.

O'Flaherty, E.J. (1983). "Toxicokinetics and Dose," presented at a workshop on toxicokinetics in the safety evaluation of chemicals sponsored by the Electric Power Research Institute in San Diego, California, November 30 - December 2.

Sichak, S.P. and O'Flaherty, E.J. (1983). Urethane kinetics and pulmonary adenoma prevalence in Swiss-Cox mice. *The Toxicologist* 3, 145.

Sichak, S.P. and O'Flaherty, E.J. (1982). The kinetics of urethane metabolism in Swiss-Cox mice. *The Toxicologist* 2, 46.

Adams, W.D., O'Flaherty, E.J., and Taylor, E. (1982). Lead exposure and renal function in rats: is the rat a good model for lead induced nephropathy? *The Toxicologist* 2, 55.

Buben, J.A. and O'Flaherty, E.J. (1982). Relationship between trichloroethylene metabolism and its hepatotoxicity. *The Toxicologist* 2, 38.

Bonewitz, R.F., Foulkes, E.C., O'Flaherty, E.J. and Hertzberg, V.S. (1982). Kinetic evidence for glucocorticoid enhancement of jejunal zinc uptake. Intern. Symposium on Biological Aspects of Metals and Metal-related Diseases. Toronto, Ontario, October 19-22.

Hammond, P.B., Hong, C.D., O'Flaherty, E.J., Lerner, S.I. and Hanenson, I.B. (1981). The rat as an animal model of lead nephropathy. Presented at the Conference on Drugs and Environmental Toxicants, Pinehurst, North Carolina, March 18-20.

Buben, J.A. and O'Flaherty, E.J. (1981). Comparison of hepatotoxicities of trichloroethylene and perchloroethylene. Abstracts, Society of Toxicology Annual Conference, San Diego, California, March.

Curriculum Vitae
Ellen J. O'Flaherty

O'Flaherty, E.J. and Hammond, P.B. (1980). Renal reabsorption of δ-amino-levulinic acid in control and lead exposed rats. Abstracts, Society of Toxicology Annual Conference, Washington, DC, March.

Hammond, P.B., O'Flaherty, E.J. and Gartside, P.S. (1979). The impact of ambient air lead on uptake and retention of lead in man - a critique of the recent literature. Presented at the International Conference, Management and Control of Heavy Metals in the Environment. London, England, September, 1979, CEP Consultants Ltd., 26 Albany St., Edinburgh, UK, pp. 93-102.

Thomas, W.C., Jr., O'Flaherty, E.J., Bell, E.H. and Stemmer, K.L. (1978). The effect of polymer pyrolysis fumes on the activity of rat hepatic cytochrome c oxidase. Abstracts, Society of Toxicology Annual Conference, San Francisco, March 13-17.

Choudhury, H., Srivastava, L., Murthy, L., O'Flahety, E.J. and Petering, H. (1974). Effects of castration and adrenalectomy on serum zinc and copper. Proc. Vth Asia and Oceania Congress Endocrinol. 1, 216.

O'Flaherty, E.J., Murthy, L. and Petering, H.G. (1974). The influence of dietary manganese and chromium on serum ceruloplasmin activity, copper and zinc in male rats. *Fed. Proc.* 33, 668.

Murthy, L., O'Flaherty, E.J. and Petering, H.G. (1971). Effect of dietary levels of copper and zinc on serum lipids in rats. Summary Abstracts, 9th International Congress on Nutrition, Mexico City, p. 136.

Letters:

Hammond, P.B., O'Flaherty, E.J. and Gartside, P.S. (1982). Impact of air-lead on blood-lead in man. (Letter to the editor). *Food and Chemical Toxicology* 20, 493.

O'Flaherty, E.J. and Dourson, M.L. (1983). Cells of origin of lung tumors in mice. (Letter to the editor). *J. Natl. Cancer Inst.* 70, 991-992.

COMMENTS ON

"MADISON COUNTY LEAD EXPOSURE STUDY, GRANITE CITY, ILLINOIS"
by
R. Kimbrough, M. LeVois, and D. Webb

AND ON THE RELATED DOCUMENTS

"COMMENTS ON MADISON COUNTY LEAD EXPOSURE STUDY,
GRANITE CITY, ILLINOIS"
by
A.H. Marcus, K. Hogan, P. White, and P. Van Leeuwen

AND

"RESPONSE TO COMMENTS OF U.S. EPA REVIEWERS
REGARDING THE GRANITE CITY LEAD STUDY DRAFT REPORT"
by
M. LeVois

AND

"PRELIMINARY ASSESSMENT OF DATA FROM THE MADISON COUNTY
LEAD STUDY AND IMPLICATIONS FOR REMEDIATION
OF LEAD-CONTAMINATED SOIL"
by
A.H. Marcus

DOCUMENTS REVIEWED BY

Ellen J. O'Flaherty
November 22, 1994

THE STUDY

Madison County Lead Exposure Study, Granite City, Illinois (hereafter referred to as *Study*) is the report of a study of environmental lead exposure and blood lead concentrations in children, youths, and adults in Granite City, Illinois, carried out by the Illinois Department of Public Health (IDPH) in late 1991. The primary hypothesis tested was whether lead in soil contributed significantly to blood lead levels in children less than 6 years old. Participants were recruited from four residential areas comprising rough concentric rings around a closed secondary lead smelter, the NL/Taracorp site. Preliminary monitoring data had indicated that soil lead concentrations decreased with distance from the smelter. The innermost area is a Superfund site. Because no suitable control residential area (e.g., similar housing stock and similar socioeconomic status of residents) could be identified, the four residential sampling areas served only to achieve a fairly representative range of soil lead values. Measured soil lead values, not sampling areas, were used in the statistical analysis.

Blood lead concentrations and the content of lead in environmental samples of dust, paint, soil, and drinking water were the key measured lead variables. Other variables, such as indicators of socioeconomic status (SES), condition of the exterior of the house, and condition of the interior paint were elicited by observation or by questionnaire.

Data analysis proceeded through a series of logical steps. Bivariate analyses were applied initially to the data from groups of children with blood lead concentrations greater than/equal to or less than 10 ug/dl and from groups of children living in regions with composite soil lead levels greater than/equal to or less than 500 ppm. The purpose of the bivariate analyses was to provide a simple first screening to eliminate variables not associated with blood lead. This preliminary data evaluation was followed by multiple regression analyses whose purposes were (1) to help identify variables that had utility in predicting blood lead concentrations, (2) to identify by means of maximum regression coefficient (R^2) improvement analysis the subset of variables with the greatest predictive utility, and (3) by means of hierarchical regression modeling, to evaluate the contribution of soil lead to blood lead and house dust lead.

The preliminary bivariate data analysis narrowed the list of potential predictor variables to lead in indoor paint, soil lead, dust lead and dust loading, distance from the NL/Taracorp site, parents' level of education, parents' income, variables related to smoking, number of hours spent outdoors by the child per day, number of baths taken per week, and the categorical variables presence/absence of air conditioning, renting/owning, condition of exterior of residence, and refinishing of the residence within the past year or of furniture within the past three months.

Stepwise regression analysis was used to select and assign level of importance to the critical predictors. The two primary predictors were found to be dust lead and distance from the smelter site, the two together accounting for 21% of the total variance in blood lead. Other predictors were the parents' level of education, number of cigarettes smoked per day, rent/own home, refinishing activities, ethnicity, dust load, age, drinking water lead concentration, distance, and number of hours of outdoor play per day. Together with dust lead and distance from the smelter site, these variables accounted for 35% of the blood lead

variance.

Hierarchical regression focused specifically on the contribution of paint and soil lead to blood lead, using a very small set of predictor variables so as not to overadjust by confounding. Water lead level, house paint lead levels, recent refinishing activities, and condition of the residence were the only potentially confounding variables introduced. House dust was not included as a potential confounder, since it was a pathway for the two primary lead sources, soil and paint. Soil lead alone was found to account for 3% of the variance in blood lead, while the condition of the house and the amount of lead in paint together accounted for about 11% of the variance in blood lead. Soil lead accounted for 6% of the dust lead variance, while indoor and outdoor paint lead levels and the condition of the residence together accounted for 26% of the variance in dust lead.

The conclusion is that at this site, soil was less important as an ultimate lead source than interior and exterior paint. Both soil lead and paint lead were found to contribute to dust lead, a pathway of exposure. Building condition was an important modifier of the contribution of paint lead to dust lead. Paint lead levels and building condition together accounted for about four times as much (26%) of the variance in dust lead as soil lead did (6%).

THE COMMENTS AND THE RESPONSE

Comments on Madison County Lead Exposure Study, Granite City, Illinois (hereafter referred to as *Comments*) is a detailed analysis of the study report. In general, many of the points raised are conventional, almost textbook recommendations for the conduct of environmental studies; however, often they miss the mark. Sometimes they do not seem to be connected with the section of *Study* being commented upon at all. The fundamental problem is that the reviewers do not seem to recognize that the study design used here was not the conventional environmental epidemiology study design with an exposed community group and a control group. In addition, a number of the points they raise had already been raised and discussed by the authors of the study report, suggesting that the reviewers did not read the report in full.

I have divided my comments on *Comments* in accordance with the structure adopted in both *Study* and *Comments*. I also refer to *Response to Comments of U.S. EPA Reviewers Regarding the Granite City Lead Study Draft Report* (hereafter referred to as *Response*).

1. IMPLEMENTATION OF STUDY DESIGN

1.1 Recruitment of Subjects

This section contains some general comments about avoidance of bias in recruiting. The stated concern that the participation rate might have been lower in areas with higher exposure and lower resident socioeconomic status is not supported by the observation that the participation rate was about equal in three of the four areas, but lower in the area with the

lowest lead exposure — a point already made and discussed in *Study*.

1.2 Omission of Pontoon Beach Subjects

Nothing would have been gained by including the small group of Pontoon Beach subjects, whose housing was entirely different from the housing in Granite City. A control group was not needed for the statistical analysis used.

1.3 Resampling of Children with Elevated Blood Lead

The logic of the first paragraph is not clear. How does bias due to inclusion of siblings follow from the "regression to the mean" problem? And what does the inclusion of some sibling measurements have to do with the conclusion that was reached by the authors, which was simply that counseling resulted in a drop in blood lead in those children whose families were counseled? Those drops were almost 50%. *Comments* notes that in other studies, children tested in winter have had lower blood lead concentrations, typically by about 30%, than children who were tested at the summertime peak, and suggests that this phenomenon invalidates the observation that counseling was followed by reductions in blood lead. In fact, *Study* discusses the seasonal fluctuation in blood lead concentrations in children in some detail, and points out that the first blood samples were taken when the time of the summer peak was already past.

The authors of *Study* made no claim that they were testing a hypothesis about the effectiveness of counseling and education in reducing blood lead. It is unnecessary for the reviewers to outline a study that could be used to test such a hypothesis, and irresponsible for them to suggest that "...the resampled children in the Madison County study are used to reach some very broad and general conclusions....". The authors have not generalized from the data from the resampled children. They have simply, and correctly, stated an observation.

2. FIELD SAMPLING AND ANALYSIS OF SAMPLES

2.1-2.3 I have no comments on these sections.

2.4 House Condition and Paint Condition

I agree with the reviewers that clearer statements of the criteria for classification of housing condition as good, fair, or poor would have been helpful. In addition, it is not always immediately clear in *Study* whether exterior or interior paint condition, or both, are included in a variable to be tested. These are minor points related to clarity of presentation, unrelated to study design or conduct.

3.STATISTICAL ANALYSIS OF DATA

3.1 Dependence on Age

It is pointed out in *Response* that blood lead models were not adjusted for age, as the reviewers mistakenly thought.

3.2 Inadequate Spatial Resolution of Demographics and Lead Exposure

The reviewers seem to have missed the point entirely here. They are still viewing the study as one in which different groups are to be compared: groups that should be identical in all respects except magnitude of lead exposure. They take issue with use of concentric geographic rings to define subject groups, but in fact subject groups were not used in the study. They remark that older housing may contain a disproportionate number of families with more than one child less than 6 years old, when it was clearly stated in the report that the study unit was the child, not the household, for most of the statistical analyses; and was also shown that outcome was not affected by using either all children in each family, or only the child with the highest or lowest blood lead concentration (*Study*, Table 13 and p. 44). The reviewers then proceed to suggest an alternate study design, one that might distinguish among sub-regions with different exposure characteristics. The purpose of this exercise is not entirely clear, but it may have been to demonstrate that the percentage of children with blood lead concentrations above 10ug/dl is not uniform throughout the study area, an observation that is interesting, but irrelevant in view of the study design. Why this is considered by the reviewers to be "absolutely vital information," as stated in the last sentence of this section, is also not clear.

The reviewers also point out in connection with this observation that "...distance alone does not describe the distribution of elevated blood lead in the study area." The authors have not claimed that it does, and again this point is irrelevant in view of the study design.

3.3 The Regression Model

Response deals quite adequately with this silly misinterpretation.

3.4 Contribution of Soil Lead to Blood Lead and to Dust Lead

Again, the authors of *Study* have explained very clearly in *Response* the failure of the reviewers in *Comments* to understand the purpose and process of hierarchical regression.

3.5 Multi-Media and Multi-Pathway Lead Exposure

Path analysis would have been one technique for dealing with these data, a technique that the authors did not choose to use. But why do the reviewers believe that, "This approach would be far more useful in identifying appropriate goals for environmental intervention."? As pointed out in *Response*, there is again evidence in this section of *Comments* that the reviewers have failed to understand hierarchical regression analysis and its

interpretation.

3.6 Individual Behavioral Variables

Response addresses this point particularly well, and sums up what I believe is the central problem in *Comments*: "...adding behavioral or other variables to a hierarchical regression model can only reduce the variance accounted for by soil. The reviewer seems to want to find some set of variables that lead to a higher simultaneous parameter estimate for soil, regardless of how little variance is explained by the individual variables, or how all of the other environmental variables are affected by the factors the reviewer wants included in a single analysis."

3.8 Multicolinearity

Another comment effectively rebutted in *Response*. It is pointed out that this comment is antithetical to the previous one!

3.9 Biases Due to Predictor Measurement

Again, a "textbook" comment that is not uniquely relevant to this study. It almost seems that the authors of *Comment* are attempting to provide a review of design difficulties and statistical uncertainties inherent to all epidemiological studies, and to associate these uncertainties with this particular study whether or not they are related to the study design and to the use of hierarchical regression analysis techniques.

4. PRESENTATION OF RESULTS

4.1 Statistical Tables

No comments.

4.2 Graphs

The graphs in Figures 2a and 2b are perfectly clear. They are not histograms.

4.3 Maps

No comments.

4.4 Confidence Intervals

This comment is adequately rebutted in *Response*.

5. INTERPRETATION AND CONCLUSIONS

1. This is a matter of interpretation.

2. Child age was *not* entered as a monotone predictor of blood lead. This criticism is inappropriate.

3. While clusters of cases representing elevated lead exposure were laboriously extracted by the reviewers from the map included in *Study*, it is not clear what the function of this exercise was. The negative association of distance with blood lead concentration was documented and reported in *Study* (pp. 38, 49).

4. The reviewers miss the point again. The *pattern* of soil lead distribution was not relevant to the study, since *actual* soil lead levels rather than radial distances were used in the analysis.

5. This comment is wrong. It is effectively rebutted in *Response*.

6. Again, this comment is effectively rebutted in *Response*.

7. The comment in *Response* is appropriate.

8. Again, the comment in *Response* is appropriate.

9. This criticism is wrong. Its gratuitous suggestion that the data analysis should be redone would be suspect if only because of the associated and wholly unsupported statement that, "The reported results are highly compatible with the causal model we proposed in Section 3.4, that lead in soil is an important indirect source of lead in blood through the soil-to-dust pathway."

THE REASSESSMENT

The report, *Preliminary Assessment of Data from the Madison County Lead Study and Implications for Remediation of Lead-Contaminated Soil*, by Allan Marcus (hereafter called *Reassessment*), is difficult to characterize. Dr. Marcus states that this report is very preliminary and that a more detailed report will be prepared that will describe the methods used in the analyses and will present a complete set of results together with the basis for his conclusions. Nonetheless, there are fundamental flaws in the analysis as it is presented here, along with the large gaps that are to be filled at a later date.

Section 1. Here, Dr. Marcus states the goals of this data reanalysis. Two of them, to provide site-specific information about relevant parameters and to evaluate the proposed soil remediation level "using this recent information," are not met, as will be detailed below.

Section 2. In Section 2, Dr. Marcus reanalyzes the data from the IDPH study. The "reanalysis" consists, first, of restatements of the fractions of the study group of children with blood lead concentrations above specified cutoff levels, starting with 10 ug/dl. These fractions are given in *Study*. It is not clear what Dr. Marcus intends to imply by repeatedly underscoring "in the study" when he refers to households or children with particular characteristics (points 2, 7, and 8). These restatements are followed by a series of graphs in which the logarithms of a number of variables such as mean blood lead concentration and environmental lead concentrations are plotted against distance from the NL/Taracorp site within 10 concentric rings around the site, each about 1/8 mile in thickness. On the basis that blood lead, soil lead, and dust lead all show similar patterns of decreasing concentration with increasing distance from the NL/Taracorp site, he concludes that both soil lead and dust lead are contributors to blood lead (point 3). However reasonable it may seem to be on the surface, such a conclusion is absolutely unjustifiable. Simple correlations cannot support such a conclusion. Following the detailed criticism in *Comments* of every aspect of the statistical methods used in *Study*, Dr. Marcus' leap of faith here is particularly egregious.

Most of the remainder of his conclusions in this section are unexceptionable, although most of them are basically restatements of the data, some of them seem to be general statements with no necessary relationship to the present data set, and some of them verge perilously close to personal opinion unsupported by data from the study ("Households in the study with the most children *and the fewest resources to cope with lead poisoning* are located closest to the NL site." (italics added)). The purpose of one other statement is unclear, however: point 9. Dr. Marcus points out that the participation rate was lower in the zone farthest from the NL/Taracorp site, and expresses concern that the sample of children may therefore not be representative of the community. In view of the fact that the four zones were not all the same size, it is not immediately clear whether this concern is justified. However, more to the point is that such a maldistribution would, if it had any effect at all, have skewed the distribution of blood lead concentrations in the direction of higher rather than lower values.

On the basis, apparently, that the linear regression relationships that were so strongly criticized in *Comments* are statistically significant and that the data set is similar to those from other EPA sites, Dr. Marcus judges that these data can be used for evaluating childhood lead exposure in Madison County.

Most troubling about this entire "reanalysis," apart from its cavalier mixing of correlations, general statements, and personal judgment, all unleavened with any statistics (although presumably these will be made available later), is that it is based entirely on the distance of concentric rings from the plant site. In *Comments*, Section 3.2, with Dr. Marcus as the first author, it is bluntly stated that, "The division of the study area into concentric rings is not defensible." Surely, what was not defensible then should not be defensible now. Unfortunately, one is left with the rather strong impression that features of the IDPH study that were judged unacceptable when they seemed to point to one conclusion have become acceptable when they are used to buttress a different conclusion.

Section 3. This analysis is little more than a running of the IEUBK model with its default parameters. Site-specific exposure data were not used. One site-specific parameter

was examined in a sensitivity analysis. The statement that, "Site-specific parameters were based on our judgment and analyses that the NL site had many points of similarity to the calibration site, Midvale,...." is disingenuous in the extreme. Midvale is a town, not an urban community, associated with a mining site, not a smelter. That the default parameters of the IEUBK model give a blood lead concentration distribution that is similar (although there is considerable discrepancy in the lower tail) to the observed distribution in the Granite City study does not imply that another set of parameters, perhaps one that was truly site-specific, would not give an equally good or even better match.

A small sensitivity analysis was carried out, varying the value of the soil-to-dust coefficient using the default value and three additional values based on "statistical analyses from study data." It is not stated what kind of statistical analysis was used. The default value of the soil-to-dust coefficient "...was judged to be appropriate, and also provided a very good fit to the child blood lead data...." In addition, the two larger values of the soil-to-dust coefficient tested, including the default value, "...are more appropriate for risk assessment, more realistic for properties of the site, and provide a good fit to the data." Since two of the three values stated to have been generated from study data were excluded by this statement, it is hard to see how the larger values are more representative of properties of the site. Curiously, however, the final suggested range of soil cleanup values *includes* the values generated by use of the two previously-excluded lower soil-to-dust coefficients, but *excludes* the values generated by use of the previously-recommended larger soil-to-dust coefficients. We are not told whether the two lower soil-to-dust coefficients also gave good fits to the distribution of children's blood lead concentrations.

The overall impression given by this entire "reanalysis" is that the recommended soil remediation level was predetermined. The "reanalysis" is superficial and careless, and bears little if any relationship to the data from the IDPH study.

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GUIDANCE MANUAL FOR THE INTEGRATED EXPOSURE UPTAKE BIOKINETIC MODEL FOR LEAD IN CHILDREN

Prepared by

THE TECHNICAL REVIEW WORKGROUP FOR LEAD

for

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U.S. ENVIRONMENTAL PROTECTION AGENCY

with Document Production Assistance from

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This document has been reviewed in accordance with U.S. Environmental Protection Agency policy and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

The Guidance Manual has been developed to assist the user in providing appropriate input to the Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead. The IEUBK Model is designed to model exposure from lead in air, water, soil, dust, diet, and paint and other sources with pharmacokinetic modeling to predict blood lead levels in children 6 months to 7 years old. This manual emphasizes the use of the IEUBK Model for estimating risks from childhood lead exposure to soil and household dust that might be encountered at CERCLA/RCRA sites, although other applications of the model are possible. The manual provides background information on environmental exposure parameters and recommends some useful approaches that allow flexibility for site-specific risk assessments, where possible. Default parameters are recommended unless there is sufficient data to characterize site-specific conditions. A separate Appendix on sampling is being developed and will be issued later. A Technical Support Document details the basis for the biokinetic parameters and equations in the IEUBK Model. In addition, EPA is continuing to compare the results of field studies with model predictions and will release these findings in a later document.

One of the proposed uses of this model will be support for the implementation of an Interim Directive of the Office of Solid Waste and Emergency Response (OSWER). This Interim Directive explains how the IEUBK Model results can be a tool for the determination of site-specific cleanup levels. In this context, the model is viewed as a predictive tool for estimating changes in blood concentrations as exposures are modified. The model is also viewed as a useful tool that should aid the Agency in making more informed choices about the concentrations of lead that might be expected to impact human health.

The development of the model has included the cooperative efforts of several EPA programs over nearly a decade. For the last three years, these efforts have been coordinated by the Technical Review Workgroup for Lead. During its development, the model has undergone review by outside scientists, and its usefulness has been evaluated by EPA staff, contractors, and other reviewers assessing site-specific risk. The current version of the IEUBK model and the Guidance Manual incorporates many of their recommendations.

The use of mathematical and statistical models for environmental risk assessment has become increasingly widespread because of the many practical difficulties encountered in controlling human exposure to toxicants with subtle and long-lasting effects. Exposure to lead during infancy and childhood increases the risk of irreversible neurobehavioral deficits

at levels of internal exposure as low as 10 to 15 μg Pb per 100 mL of blood (10 to 15 μg/dL). Lead has many known sources, and many pathways from its environmental sources into the child's body (U.S. Environmental Protection Agency, 1986). The Environmental Protection Agency has long been interested in methods for relating environmental lead concentrations to blood lead concentrations in children. Earlier approaches based on statistical correlations provided essential information on the existence and magnitude of childhood lead uptake from persistent exposure to different environmental sources, including lead in air, diet, drinking water, soil, dust, and lead-based paint. Unfortunately, these statistical relationships are limited in their ability to estimate the effects of alternative lead abatement methods that change pathways as well as sources.

In 1985 the EPA Office of Air Quality Planning and Standards began to develop an alternative approach for estimating the effectiveness of alternative National Ambient Air Quality Standards for lead, particularly around point sources of air lead emissions such as smelters. This was a computer simulation model with two components: (1) a model of the biokinetics of lead distribution and elimination whose parameters vary with the child's age, and (2) a multi-source and multi-media lead exposure model in which air lead concentrations change over time. The biokinetic model was based on studies at New York University by Naomi Harley, Theodore Kneip, and Peter Mallon. The U.S. Environmental Protection Agency Clean Air Science Advisory Committee (CASAC) reviewed and found acceptable the OAQPS staff report documenting the model in 1989. A subsequent OAQPS staff paper reviewing the National Ambient Air Quality Standard for Lead, which included results of applying the model to point sources of air lead such as smelters and battery plants, was also evaluated by CASAC in 1990 (U.S. Environmental Protection Agency, 1990B).

Those who had been involved in developing the lead model then received a large and growing number of requests on applications of the model in a wide variety of other contexts not originally intended for model use. The largest number of these requests involved the use of the model to estimate the effects of soil lead abatement at Superfund sites.

The air model was further developed to include enhancements in absorption and biokinetics. In November, 1991, the Indoor Air Quality and Total Human Exposure Committee (IAQTHEC) of EPA's Science Advisory Board (SAB) reviewed the Uptake Biokinetic Model for Lead (version 0.4) and evaluated its use in assessing total lead exposures and in aiding in developing soil cleanup levels at residential CERCLA/RCRA sites. The Committee's Report was transmitted to EPA Administrator William K. Reilly in March, 1992. The Committee concluded that while refinements in the detailed specifications

of the model would be needed, the approach followed in developing the model is sound. The Committee stated that the model can effectively be applied for many current needs even as it continues to undergo refinement for other applications, based upon experience gained in its use.

The Committee was concerned that the reliability of the results obtained using the model is very much dependent on the selection of the various coefficients and default values that were used. In particular, the Committee identified the need for guidance on the "proper" geometric standard deviation (GSD) and the use of default values for other parameters. In addition to these general comments, specific comments were included in the Report. The comments of the SAB and other reviewers have been considered in this revision of the Guidance Manual.

Since the SAB review, EPA has further refined the model. The four main components of the current IEUBK model are: (1) an exposure model that relates environmental lead concentrations to age-dependent intake of lead into the gastrointestinal tract; (2) an absorption model that relates lead intake into the gastrointestinal tract and lead uptake into the blood; (3) a biokinetic model that relates lead uptake in the blood to the concentrations of lead in several organ and tissue compartments; and (4) a model for uncertainty in exposure and for population variability in absorption and biokinetics. A Technical Support Document that details the selection of parameters and equations in the model is available.

As with any multicompartmental model, pools in the compartmental analysis can be identified with specific organs or organ systems only if biological concentrations of the compartments are known. For some compartments, the biological concentrations have been measured at a number of time points so that the movement of lead from one compartment to another can be estimated. The biokinetic and absorption components of the model, however, are not observed directly but are inferred from accessible data.

In developing the IEUBK Model, EPA has learned much from "real world" comparisons of blood lead and predicted values—not only that the model works, but also that it can be made to work better. Guidance on the appropriate use of the model is based on our experiences, where possible, and on the experiences of many users and reviewers of the model. Many of the most useful parts of the Guidance Manual have been suggested by these reviewers.

While the model has been used to support the NAAQS for Lead, the Clean Water Act national regulations, and several other regulatory and enforcement issues, EPA is continuing its validation of the IEUBK Model with detailed evaluation of additional data collected from different types of sites. Comparison of predicted and empirical blood lead concentrations will be described in the Field Study Data Set Comparisons Document described in Section 1.2.2.

Although EPA is releasing version 0.99d of the IEUBK Model to ensure consistent application among users, the Agency will continue to evaluate the results of validation exercises and different applications of the model. The Environmental Protection Agency will determine periodically whether refinements to the model are warranted, considering scientific advancements and the development of alternative approaches.

The Environmental Protection Agency welcomes the suggestions of those using the IEUBK model. Questions regarding the site-specific application of the IEUBK Model should be raised with the appropriate Regional Toxics Integration Coordinator. Comments on the technical content of the manual or suggestions for its improvement may be brought to the attention of the Technical Review Workgroup for Lead, whose current addresses are listed on page xxi.

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GLOSSARY OF MODEL TERMS

Absorbed dose - The amount of a substance penetrating an absorption barrier (the exchange boundaries) of an organism, via either physical or biological processes.

Absorption barrier - Any of the exchange barriers of the body that allow differential transport of various substances across a boundary. Examples of absorption barriers are the skin, lung tissue, and gastrointestinal tract wall.

Accuracy - The measure of the correctness of data, as given by the difference between the measured value and the true or standard value.

Ambient - Surrounding conditions.

Ambient measurement - The measurement (usually of the concentration of a chemical or pollutant) taken in an ambient medium, normally with the intent of relating the measured value to the exposure of an organism that contacts that medium.

Ambient medium - One of the basic categories of material surrounding or contacting an organism (e.g., outdoor air, indoor air, water, or soil) through which chemicals or pollutants can move and reach the organism. (See biological medium, environmental medium.)

Arithmetic mean - The sum of all the measurements in a data set divided by the number of measurements in the data set.

Background level (environmental) - The concentration of substance in a defined control area during a fixed period of time before, during or after a data gathering operation.

Bias - A systematic error inherent in a method or caused by some feature of the measurement system.

Bioavailability - The fraction of intake at a portal of entry into the body (lung, gut, skin) that enters the blood. Bioavailability is typically a function of chemical properties, physical state of the material that an organism ingests or inhales, and the ability of the individual organism to physiologically absorb the chemical. The absorption rate varies widely by type of substance and can greatly influence the toxicity of lead over that acute timeframe.

Biokinetics - processes affecting the movement of molecules from one internal body compartment to another, including elimination from the body.

Biological measurement - A measurement taken in a biological medium. For the purpose of exposure assessment via reconstruction of dose, the measurement is usually of the concentration of a chemical/metabolite or the status of a biomarker, normally with the intent of relating the measured value to the internal dose of a chemical at some time in the past.

(Biological measurements are also taken for purposes of monitoring health status and predicting effects of exposure). (See ambient measurement.)

Biological medium - One of the major categories of material within an organism (e.g., blood, adipose tissue, or breath) through which chemicals can move, be stored, or be biologically, physically, or chemically transformed. (See ambient medium, environmental medium.)

Body burden - The amount of a particular chemical stored in the body at a particular time, especially a potentially toxic chemical in the body as a result of exposure. Body burdens can be the result of long term or short term storage, for example, the amount of a metal in bone, the amount of a lipophilic substance such as PCB in adipose tissue, or the amount of carbon monoxide (as carboxyhemoglobin) in the blood.

Comparability - The ability to describe likenesses and differences in the quality and relevance of two or more data sets.

Compartment - A distinct anatomical organ, tissue, fluid pool, or group of tissues within the body that are regarded as "kinetically homogeneous."

Dose - The amount of a substance available for interaction with metabolic processes or biologically significant receptors after crossing the outer boundary of an organism. The potential dose is the amount ingested, inhaled, or applied to the skin. The applied dose is the amount of a substance presented to an absorption barrier and available for absorption (although not necessarily having yet crossed the outer boundary of the organism). The absorbed dose is the amount crossing a specific absorption barrier (e.g., the exchange boundaries of skin, lung, and digestive tract) through uptake processes; internal dose is a more general term denoting the amount absorbed, without respect to specific absorption barriers or exchange boundaries. The amount of the chemical available for interaction by any particular organ or cell is termed the delivered dose for that organ or cell.

Environmental medium - One of the major categories of material found in the physical environment that surrounds or contacts organisms (e.g., surface water, ground water, soil, or air) and through which chemicals or pollutants can move and reach the organisms. (See ambient medium, biological medium.)

Exposure - Contact of a chemical, physical, or biological agent with the outer boundary of an organism. Exposure is quantified as the concentration of the agent in the medium in contact integrated over the time duration of that contact.

Exposure pathway - The physical course a chemical or pollutant takes from the source to the organism exposed.

Exposure route - The way a chemical or pollutant enters an organism after contact (e.g., by ingestion, inhalation, or dermal absorption).

Exposure scenario - A set of facts, assumptions, and inferences about how exposure takes place that aids the exposure assessor in evaluating, estimating, or quantifying exposures.

Geometric mean - The nth root of the product of n values. Also, the exponential function of the mean or expected value of the natural logarithm of a variable.

Geometric standard deviation (GSD) - The exponential function of the standard deviation of the natural logarithm of a variable.

Guidelines - Principles and procedures to set basic requirements for general limits of acceptability for assessments.

Intake - The process by which a substance crosses the outer boundary of an organism without passing an absorption barrier (e.g., through ingestion or inhalation). (See also "potential dose").

Internal dose - The amount of a substance penetrating across the absorption barriers (the exchange boundaries) or an organism, via either physical or biological processes.

Matrix - A specific type of medium (e.g., surface water, drinking water) in which the analyte of interest may be contained.

Median value - The value in a measurement data set such that half the measured values are greater and half are less.

Monte Carlo technique - A repeated random sampling from the distribution of values for each of the parameters in a generic (exposure or dose) equation to derive an estimate of the distribution of (exposures or doses in) the population.

Pathway - The physical course a chemical or pollutant takes from the source to the organism exposed.

Pharmacokinetics - The study of the time course of absorption, distribution, metabolism, and excretion of a foreign substance (e.g., a drug or pollutant) in an organism's body.

Potential dose - The amount of a chemical contained in material ingested, air breathed, or bulk material applied to the skin.

Precision - A measure of the reproducibility of a measured value under a given set of conditions.

Probability samples - Samples selected from a statistical population such that each sample has a known probability of being selected.

Random samples - Samples selected from a statistical population such that each sample has an equal probability of being selected.

Range - The difference between the largest and smallest values in a measurement data set.

Reasonable worst case exposure or risk range - The lower portion of the "high end" of the exposure, dose or risk distribution. The reasonable worst case conceptually should be targeted at above the 90th percentile in the distribution, but below about the 98th percentile ("maximum exposure or risk range").

Representativeness - The degree to which a sample is, or samples are, characteristic of the whole medium, exposure, or dose for which the samples are being used to make inferences.

Risk - The probability of deleterious health or environmental effects.

Route - The way a chemical or pollutant enters an organism after contact (e.g., by ingestion, inhalation, or dermal absorption).

Sample - A small part of something designed to show the nature or quality of the whole. Exposure-related measurements are usually samples of environmental or ambient media, exposures of a small subset of a population for a short time, or biological samples, all for the purpose of inferring the nature and quality of parameters important to evaluating exposure.

Scenario evaluation - An approach to quantifying exposure by measurement or estimation of both the amount of a substance contracted, and the frequency/duration of contact, and subsequently linking these together to estimate exposure or dose.

Structural Equations Model - A statistical model of a process in which several regression equations are solved simultaneously, and outputs or responses from one equation may be used as inputs or predictors in another equation. Useful in pathway modeling.

Surrogate data - Substitute data or measurements on one substance used to estimate analogous or corresponding values of another substance.

Uptake - The process by which a substance crosses an absorption barrier and is absorbed into the body.

1. BEFORE YOU START

1.1 BACKGROUND: PURPOSE AND DEVELOPMENT OF THE MODEL

The Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children is a stand alone, PC compatible software package. It allows the user to estimate, for a hypothetical child or population of children, a plausible distribution of blood lead concentrations centered on the geometric mean blood lead concentration predicted by the model from available information about children's exposure to lead. From this distribution, the model calculates the probability that children's blood lead concentrations will exceed the user selected level of concern (default 10 μg/dL). The user can then explore an array of possible changes in exposure media that would reduce the probability that blood lead concentrations would be above this level of concern.

The model should be viewed as a tool for making rapid calculations and recalculations of an extremely complex set of equations that includes scores of exposure, uptake, and biokinetic parameters. This Guidance Manual concisely describes key features of the conceptual underpinnings of the IEUBK model, its evolution and development, its capabilities, and its limitations. The Manual then goes on to offer guidance on the use of the model as a risk assessment tool while cautioning against a number of possible misapplications of the model. A detailed description of the equations and parameters used in the model is provided in the Technical Support Document: Parameters and Equations Used in the Integrated Exposure Uptake Biokinetic Model for Lead in Children (a companion document to this Guidance Manual).

1.1.1 Description of the Model

The IEUBK Model is a simulation model. As a risk assessment tool, it can be a useful component of remediation strategies for lead in the human environment. The simulation of childhood lead exposure and retention is only one part of the risk assessment process. It is important to note that the model alone does not determine the level of cleanup required for a specific site. Rather, it predicts the likely blood lead distribution for children given the exposure to lead at that site, and the probability that children exposed to lead in that environment will have blood lead concentrations exceeding a health-based level of concern.

Blood lead concentrations are not only indicators of recent exposure, but also are the most widely used index of internal lead body burdens associated with potential health effects. Health effects of concern have been determined to be associated with childhood blood lead concentrations at or below 10 μg/dL (U.S. Environmental Protection Agency, 1986, 1990; CDC, 1991). The probability that children will have blood lead levels exceeding this level of concern is an important consideration for a risk assessor in compiling and evaluating all information applicable to a site to enable remediation decisions.

The IEUBK model can be applied at several different scales of application, but the interpretation of the model output and the form of the model or subsequent risk estimates is different for each application. In most uses of the model, a site is a spatial domain that is appropriate for remediation decisions, typically a residential yard with a single housing unit, or an equivalent area for multi-unit buildings or for undeveloped lots. The home and its surrounding yard is the basic unit for risk analysis because lead exposure for pre-school children commonly occurs within this domain. In Sections 1.4.4.2 and 4.2 we will describe an array of applications of the IEUBK model based on aggregating clusters of sites. The array is:

A: One location
- A1: one living unit, one child;
- A2: one living unit, more than one child;
- A3: more than one living unit, more than one child, homogeneous media concentrations;

B: Multiple locations, one neighborhood, homogeneous media concentrations

C: Multiple locations, one neighborhood, heterogeneous media concentrations;

D: Multiple locations, more than one neighborhood, heterogeneous media concentrations;

In category A, risk is calculated as the probability that, in a single child at a single site with the specified exposure scenario, the child's blood lead concentration will exceed the level of concern. The probability distribution describes the likely variability in blood lead for a child with a given exposure scenario. The best single-number prediction of blood lead concentration is the geometric mean of the distribution of blood lead concentrations that may occur for a child with the specified exposure scenario. This single-child assessment is used to evaluate remediation options on a house-by-house or yard-by-yard basis.

In categories B, C, and D, a frequency distribution of the individual risk of exceeding a blood lead level of concern is obtained. The percentage of children in multiple sites that are likely to have a blood lead concentration exceeding the level of concern can then be calculated. For category B, where all children of the same age have the same exposure scenario, this can be done with a single run of the IEUBK model. For categories C and D, where distinct subgroups have different exposure scenarios, risk must be calculated by aggregating the results from a number of model runs. Risk estimation for more than one neighborhood, for category D, has the added complication that a variety of model parameters may differ between neighborhoods, and within each neighborhood. Therefore, environmental lead concentrations may differ between neighborhood subgroups.

1.1.2 Simulation of Childhood Lead Exposure and Retention

Lead is a naturally occurring nonnutrient metal that follows environmental pathways similar to those of nutrient metals such as calcium. In the human environment, these pathways or routes of exposure transfer lead from sources such as food, drinking water, air, soil, and dust, to the human body by means of ingestion or inhalation. There are important analogies to be made between lead and calcium that contribute to our understanding of the biological behavior of lead. These analogies have aided in the formulation of the lead model. In particular, the nature of gut absorption of lead and calcium may be similar. Childhood growth and development of bone and soft tissue which require calcium influence the uptake of environmental lead from the gut. In addition to similarities in absorption, both lead and calcium are stored in quantity and subsequently released from bone tissue.

Shown conceptually on Figure 1-1, inhaled or ingested lead is absorbed through the lungs or gut into the blood stream where it is transferred to body tissues, including bone tissues. After a period of time, this lead returns to the blood stream where it is transferred to other tissues or eliminated with urine. Lead may also be eliminated from the body with sweat, hair or sloughed epidermal tissue, or it may be transferred through the liver and bile duct back to the gut where it passes out of the body with feces.

In Figure 1-1, the oval shapes show environmental lead media, and some of the pathways between them. The large rectangle shows the compartment that is central to lead distribution in the child, the blood plasma pool and associated extra-cellular fluid. Each lower rectangle shows a compartment in the child's body where lead may be retained. The excretion of lead from the body is shown by the circles.

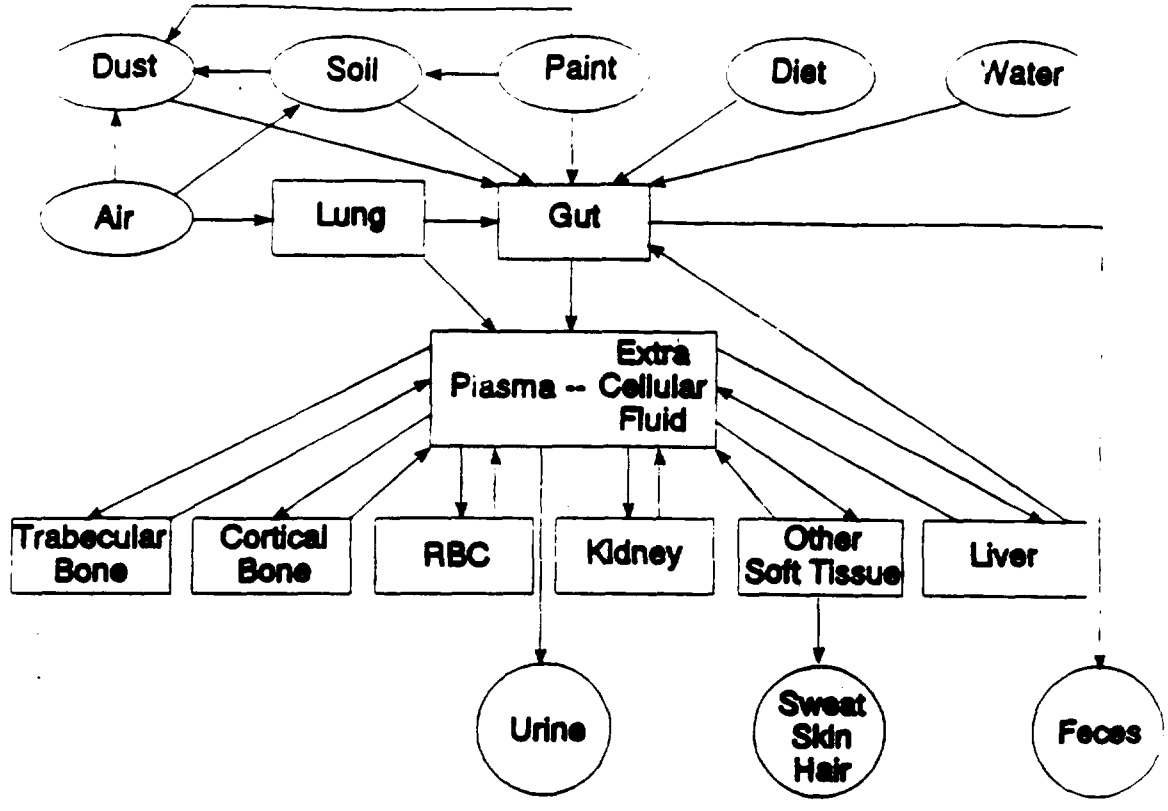


Figure 1-1. Conceptual diagram of the movement of environmental lead into and through the human body. The oval shapes show environmental media and the pathways of uptake. The large rectangle is the blood plasma compartment central to the distribution of lead in the body.

The foundation of the present IEUBK model is the construction of a detailed and thorough exposure scenario for children aged 0 to 84 months that can be adjusted to match the exposure of any child. The user starts with exposure information specific to these children and accepts generalized assumptions about any additional information required to complete the exposure scenario. The site-specific information usually consists of environmental media concentrations such as soil lead concentrations.

The model inserts default values whenever site-specific information is not used. The default values (e.g., dietary lead concentrations and consumption values) are typical of a child's environment in the sense that they are broad-based estimates of the expected

environment of a child. These default values are not necessarily appropriate for every site and should be reviewed by the user for every site-specific application.

This model uses standard age-weighted exposure parameters for consumption of food, drinking water, soil, and dust, and inhalation of air, matched with site-specific concentrations of lead in these media, to estimate exposure for the child. The model simulations represent chronic exposure and do not incorporate the variability in consumption patterns and media concentrations on a daily or seasonal basis. The model includes continuous growth of the child and simulates the changing environment of the child on a yearly basis. In theory, the exposure component of the model would apply to a single child or to any number of children with the same lead exposure scenario. With the proper substitution for media concentrations, the exposure component (but not the biokinetic component) would also apply to any other substance with sources and pathways of exposure similar to lead.

The model simulates lead uptake, distribution within the body, and elimination of lead from the body. The uptake portion of the model takes into consideration two mechanisms of absorption of lead in the digestive tract: saturable and non-saturable. Elimination of lead is modeled through several routes: urine, gastro-intestinal excretion, and sloughing of epidermal tissue, including hair and nails.

1.1.3 Historical Evolution from Slope Factor Models to the IEUBK Model

An explicit mathematical method for estimating the likely risk of elevated blood lead concentrations in young children has previously been used by the Environmental Protection Agency as one of its tools for developing the National Ambient Air Quality Standard for Lead and the National Primary Drinking Water Regulation for Lead. The method has historically been based mainly on an estimation of relationships between lead concentrations in children's blood and lead concentrations in specific individual environmental media such as air, water, soil and dust, based on empirical observations derived from experimentally controlled human exposure, animal toxicological studies, and epidemiological analyses. Such relationships also provide a basis for estimating the probability that elevated blood lead concentrations exceed a level of concern due to exposure to environmental lead in these media.

A mathematical approach of this type was used to evaluate potential alternative air lead standards based on health effects criteria (U.S. Environmental Protection Agency, 1977,

1978, 1989a). The relationship between blood lead and lead in environmental media was estimated statistically, both for adults and children (U.S. Environmental Protection Agency, 1986, 1989a). While the relationship was somewhat non-linear at blood lead concentrations above about 40 μg/dL in adults and 30 μg/dL in children, it was nearly linear at lower blood lead concentrations of interest. The relationship between blood lead and environmental lead concentrations in different media (air, water, soil, dust, food) was estimated using a model linear in lead concentrations. The linear regression coefficients between blood lead and lead in each of the environmental media have since become known as the slope factors for the media.

As more evidence has become available, it has become clear that these slope factors can not be regarded as universal constants that are the same everywhere, for all children at all sites. Some of the problems involved in the use of slope factors have been discussed by the U.S. Environmental Protection Agency (1989a) and by Brunekreef et al. (1984). In the development of improved lead models (U.S. Environmental Protection Agency 1986, 1989a), the following points were discussed:

(1) Slope factors are a function of many factors: media ingestion rates; bioavailability and absorption of lead from the medium; and biological kinetics of lead retention and elimination in the child. Biological and physical differences between sites and study populations cannot be incorporated explicitly and quantitatively into regression slope factors from different studies.

(2) Slope factors for a single medium, such as lead in air or lead in soil, may provide only a very incomplete picture of total lead exposure from a particular source, even if the source is identified with the medium. A single medium such as household dust may contain lead from many sources, and lead from a single source such as exterior lead-based paint may contribute to several exposure media pathways to the child.

Therefore, in 1985, the EPA Office of Air Quality Planning and Standards (OAQPS) initiated a project that would allow the calculation of blood lead concentrations in children exposed to differing arrays of concentrations of lead in air, soil, and dust. This model, called the Uptake/Biokinetic (or UBK) model for lead, was a computer simulation model based on the biokinetic model for lead in children developed by N. Harley and T. Kneip (1985). The biokinetic parameters for the UBK model were extrapolated from long-term feeding studies on infant and juvenile baboons (Mallon, 1983), autopsy data on human children, human infant feeding studies, and other sources. The exposure model that was

coupled to the biokinetic model was developed by OAQPS. Model calibration and validation was done using data from the 1983 EPA/CDC/Montana study on children in East Helena, Montana, who lived in the vicinity of a large primary lead smelter. The modeling approach was reviewed and approved by EPA's Clean Air Science Advisory Committee (CASAC) in 1990.

The overall framework of both the UBK and IEUBK models is shown in Figure 1-2. The oval shapes show environmental lead concentrations and the funnel-shaped symbols show lead intake from the environment at the portals of entry, the lung and the gut. These are the exposure/intake components of the IEUBK model. The next large rectangle shows the gut not only as the main portal of entry for lead from most exposure media, but also as the site for key absorption/uptake components of the IEUBK model for the evaluation of lead from soil, dust, diet, and drinking water. The very large rectangle shows the child's blood lead, partitioned into plasma-extracellular fluid and red blood cells. The two boxes to the right of the blood lead pool sketch the bone and soft tissue pools, and the elimination pathways are shown as circles. The right-hand box shows the blood lead concentration in the child, and the subdivisions show the estimated contribution of each medium to the child's blood lead concentration. In the example in Figure 1-2, we have assumed that all external lead media have been used in the IEUBK model, as have all internal lead sources. There is no unattributable component called "background". The attribution of specific fractions of blood lead to uptake from specific media is not as subject to statistical artifacts, since pathways from soil lead and air lead to dust lead are also included in the IEUBK model.

In all particulars, the present version of the model, the IEUBK model, may be considered an enhancement and extension of the UBK model. Theoretically, in situations where the child has constant long-term or chronic lead exposure, both the slope factor approach and the UBK model (now the IEUBK model) should produce similar results when sufficient data exist to correctly characterize lead exposure, absorption, and biokinetics.

The IEUBK Model addresses three emerging paradigms of environmental risk assessment.

(1) Assessments that recognize the multimedia nature of exposures to environmental toxicants are a significant improvement in assessing health risks. Assessments restricted to single pathways of exposure can overlook situations where integrated multimedia exposures are high enough to trigger health concerns. The lead model is structured to integrate exposures occurring through air, water, food, soil, and dust in estimating the blood lead levels in children in realistic environmental settings.

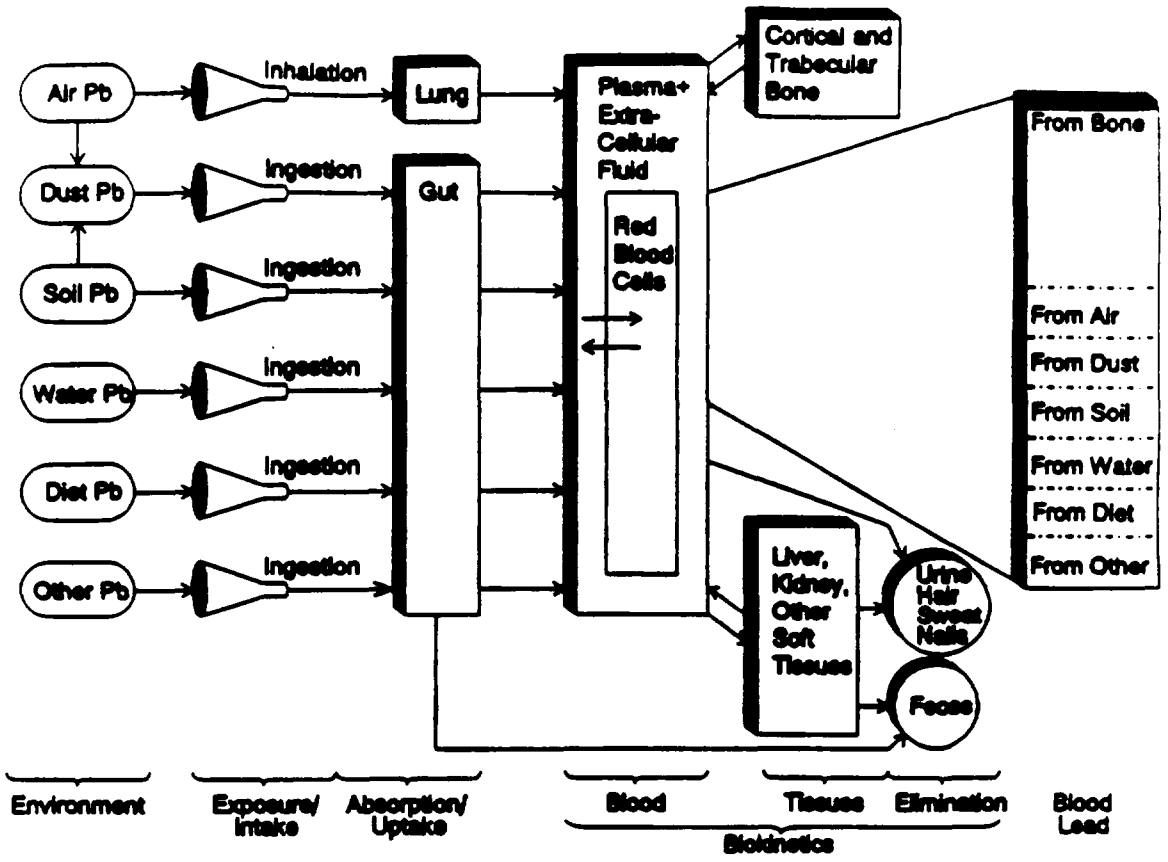


Figure 1-2. Components of the IEUBK Model, showing environmental exposure sources and pathways, absorption compartments, critical body tissue compartments, and elimination pathways.

(2) Pharmacokinetic information can strengthen the validity of environmental health assessments in comparison with more traditional methods that address only external dose or intake of a compound. Internal measures of dose that are pertinent to the biological effects exerted by a compound form an improved metric for risk assessment. The IEUBK estimates of blood lead concentrations as an internal indicator of potential health risk are based on pharmacokinetic modeling of lead absorption, transport, redistribution, and elimination.

(3) Environmental assessments need to address the substantial variability in exposure and risk resulting from these factors. Single point estimates of exposure or risk are of limited utility. Individuals differ in their surroundings, behavior, and physiological status. The Lead Model addresses variability through the estimation of probability distributions of blood lead levels for children exposed to similar environmental

concentrations of lead. Through systematic application of the model, data on the variability of levels of environmental lead contamination can be translated into estimates of the distribution of blood lead levels within populations of children.

1.1.4 Using the IEUBK Model for Risk Estimation

The IEUBK Model for lead is designed to facilitate: (a) rapid delineation of the relationship between environmental lead and blood lead in children; and (b) calculation of the risk of elevated blood lead (i.e., the probability of a given child or a group of children having blood lead concentrations exceeding a specified level of concern). As such, the IEUBK Model provides a tool for site-specific risk assessment for young children exposed to lead from different media and through different pathways in their environment, with particular emphasis on lead in air, water, soil, and household dust. Many other applications are possible. The intended applications of the IEUBK model are to:

(1) Provide a summary of children's long-term, primarily residential, exposure to lead;

(2) Provide a best estimate of the geometric mean blood lead concentration for a typical child aged 6 to 84 months, assumed to reside at a given residence;

(3) Provide a basis for estimating the risk of elevated blood lead (i.e., for exceeding a designated blood lead concentration of concern) for a hypothetical child of specified age with given site-specific residential lead exposure;

(4) Provide a basis for estimating the risk of elevated blood lead concentrations among early pediatric populations in a given neighborhood by aggregating the individual residential risk estimates;

(5) Predict likely changes in risk of elevated blood lead concentrations from exposure to soil, dust, water, or air lead following abatement actions designed to reduce exposure levels from one or more environmental media;

(6) Provide assistance in determining appropriate soil or dust lead target cleanup levels at specific residential sites;

(7) Provide assistance in estimating blood lead concentrations associated with soil or dust lead concentrations at undeveloped residential sites that may be developed in the future.

Each of these applications is discussed in more detail in Chapters 2, 4, and 5. The IEUBK model has been used for many purposes in addition to those for which it was originally intended. We are sure that the IEUBK model will continue to be used in many unintended and unexpected applications, just like any other new tool that has multiple uses. Some of these new applications are valid, others are demonstrably invalid, and the validity of many applications is simply unknown.

The risk estimates are calculated for a hypothetical child or a hypothetical population of children who could be occupying the specific household at the time of the measurements or at some future time. The IEUBK model can therefore be used to estimate the risk of elevated blood lead even when there are no children currently living at a house, or if there exist only environmental lead data for the dwelling unit. The model does not require that a neighborhood or community blood lead study be carried out. The user should be aware that a site-specific risk assessment requires site-specific soil and dust concentrations, and some of the absorption parameters may depend on specific characteristics of the soil and dust at the site. The IEUBK model accepts user inputs for site-specific differences in bioavailability of lead in different media, and site-specific differences in environmental lead pathways for different lead sources.

1.1.5 Validation of the IEUBK Model

What does it mean to say that a computer simulation model is "valid"? In general, we interpret this to mean that:

- the model is biologically and physically plausible and incorporates the best available empirical data on parameters;
- the model uses numerically accurate algorithms and the accuracy of the computer codes for these algorithms has been verified;
- the model provides some satisfactory empirical comparisons of model output with real-world data.

We believe that the scientific basis and computational correctness of the IEUBK Model is sound, and that the IEUBK model provides valid prediction of observed blood lead

concentrations from representive populations of children with typical exposure. The empirical comparisons in which there are differences between observed and predicted blood lead concentrations underscore the importance of valid exposure scenarios as input. They also show the importance of valid blood lead data from truly representative population sampling methods when interpreting these empirical comparisons.

1.1.5.1 The Model Is Biologically and Physically Plausible

The parameters and equations used in the model are documented in the Technical Support Document: Parameters and Equations Used in the Integrated Exposure Uptake Biokinetic Model for Lead in Children. The exposure model component is based on data for human children in most instances, with lead exposures that are characteristic of children in the U.S. since about 1980. The ingestion parameters are based on surveys for drinking water and tap water (Ershow and Cantor, 1989), market basket estimates of dietary intake (Pennington, 1983; Gartrell, 1986), and on observational studies of soil and dust ingestion for children in the U.S. (Binder et al., 1986; Calabrese et al., 1989, 1992a,b, 1993; Davis et al., 1990). While these studies have not resolved all of the uncertainty in childhood lead exposure, especially from sources such as lead-based paint, they have provided a much more realistic basis for quantitative modeling. The exposure component of the IEUBK model extends the UBK model assumptions (U.S. Environmental Protection Agency, 1989a) that have been reviewed by CASAC (1990).

An absorption component was developed for the IEUBK model based on evidence discussed in Section 4.1. This evidence includes in vivo data in infant and juvenile baboons and human infants whose intake of lead is observed and known (Mallon, 1983; Sherlock and Quinn, 1986). The model has two modes for absorption, saturable and non-saturable. In the non-saturable mode, absorption of lead is a constant fraction of the total lead ingested for a specific medium. The saturable mode follows the Michaelis-Menten kinetics for saturable absorption as proposed by Aungst and Fung (1981). Development of the algorithm is also based on data from lead balance and feeding studies in human infants and children (Alexander, 1974a,b; Ryu et al., 1983, 1985; Ziegler et al., 1978).

The compartmental structure of the earlier biokinetic model is based on compartmental models for lead in adults as discussed in detail in the Air Quality Criteria Document for Lead (U.S. Environmental Protection Agency, 1986). The model was verified and extended based on studies in infant and juvenile baboons (Mallon, 1983) whose age (5 to 26 months) and size (2.5 to 6 kg) are only slightly smaller than those of human children. The biokinetic distribution and elimination parameters use ratios of lead concentrations in tissues and blood

following chronic exposure. The ratios of lead concentrations in tissues of human children from autopsy data (Barry, 1975, 1981) were used to adjust the baboon's biokinetic distribution parameters to human infants and children (Harley and Kneip, 1985). The biokinetic parameters for baboons were re-estimated using the compartmental structure of the current IEUBK model (Marcus, 1992). The tissue-to-blood concentration ratios from the human child autopsy data were incorporated in the IEUBK model, assuring complete consistency with the best available data.

1.1.5.2 The Model Is Computationally Accurate

The IEUBK model uses a fast and accurate one-step numerical integration method known as 'backward Euler', with user-adjustable time steps to verify numerical accuracy of the solution. Coding of the model equations was verified by a separate recoding of the model in another programming language. Independent code verification will be described in forthcoming Technical Memoranda (see Section 1.2.2).

1.1.5.3 Empirical Comparisons of the Model

Comparison of the IEUBK model output with empirical human blood lead data has two requirements. The first requirement is that the child's total lead exposure is completely and accurately characterized by the empirical data, including site-specific data on environmental lead concentration, media ingestion, and bioavailability. The second requirement is that the blood lead data from the field study are accurate and typical for that exposure scenario. A typical child may not have the exposure described by the measured and default parameters of the model, or a child may also respond atypically to the measured and default parameters. The solution is to find the correct set of parameters (measured or site-specific alternatives to default) that describes the child's site-specific exposure or response to exposure.

Environmental lead concentrations and blood lead measurements are subject to measurement errors such as repeat sampling variability and analytical error. Without careful attention to quality assurance/quality control (QA/QC) procedures, there may be systematic biases in blood lead measurements. The results of the blood lead field study may also differ from the model predictions for typical children if the blood lead sample is not representative of the population being sampled.

Validation by empirical comparisons with paired data sets of good quality is an ongoing process. In earlier versions of the model, empirical comparisons indicated satisfactory agreement between observed and predicted blood lead concentrations. Several data sets have been identificed that are of adequate data quality for evaluating the validity of the IEUBK

Model, and more data sets are expected to become available in the future. The Field Study Data Set Comparisons document referred to in Section 1.2.2 will discuss the results of these analyses. Comparisons of empirical data with the IEUBK model require appropriate site-specific exposure scenarios, valid assumptions about bioavailability, and demonstrated representativeness of the sample of children recruited into the study in relation to the target population from which they were drawn.

Our preliminary analyses of several data sets so far indicate that the model satisfactorily predicts blood lead concentrations for the overall sample populations in specific neighborhoods. Further analyses will be needed to determine if empirical comparisons are as strong for subpopulations defined by factors such as differences in age, differences in contact or behavior that affected the amount of soil ingested, suspected or possible differences in bioavailability, differences in contribution of soil to household dust, and identifiable biases in recruitment of children. More extensive evaluation of these data sets will be described in the Field Study Data Set Comparisons document described in Section 1.2.2.

Careful determinations should be made by users with regard to how well default values specified by this manual for key exposure and demographic parameters apply to the particular sample of children (or subpopulations) being evaluated. Appropriate adjustments made in pertinent default values may notably improve the fit of the model to empirical data. We caution the user not to arbitrarily select alternate values for the default parameters, but rather to obtain site specific or population specific data on important parameters.

1.2 ORGANIZATION OF THE MANUAL

1.2.1 Increasing Levels of Guidance and Technical Assistance

This manual is designed to provide you with the information you need at several levels of detail. The further you read into manual the more specific guidance you will find for using the model. By the time you have finished reading Chapter 1, you should have a general understanding of how the model works and what it can do. You may want to install the model and then work your way through Chapter 2 as you become more familiar with each feature of the model. Instructions for installing the model are found in Section 2.4.

As you explore the various features of the model, you will become familiar with the menus and their options. An overview of the menu system is in Section 2.1, and a detailed

description of these menus can be found in Section 2.2. This is the section that the novice user will want to follow closely. In a guided tour through the menu system, you will find that each menu option becomes a part of the process of constructing a model "run," and that these runs may be as simple as determining the blood lead concentration using only default exposure conditions, or as complicated as neighborhood risk estimation calculated as the sum of individual risks. Many of these options were suggested by comments received during the extensive review of drafts of this Guidance Manual.

As you begin to apply the model to a specific risk assessment situation, you will find that Section 2.3 contains detailed recommendations for building an exposure scenario. This section also contains a helpful worksheet for planning model runs. Follow this section closely, as it contains many helpful suggestions on the appropriate use of the model, as well as warnings of improper applications. In Chapter 4, you will find a detailed discussion on assessing the relationship between soil/dust lead and blood lead. This chapter also describes the biokinetics of the model and specific issues in the use of the model for the ingestion of paint chips. If you need more help, turn to Chapter 5, where several specific examples are available to guide you through some of the more complicated procedures. As you become more experienced, you will find Chapter 3 a quick and ready reference to the various menu options. This chapter also contains a comprehensive review of default parameters.

1.2.2 Additional Documentation

Additional technical documents are or soon will be available to supplement the IEUBK Model and this Guidance Manual. These are:

- Technical Support Document: Parameters and Equations Used in the Integrated Exposure Uptake Biokinetic Model for Lead in Children—a description and documentation of all equations and parameters in the model;
- Field Study Data Set Comparisons—a description of several validation exercises that have been or will shortly be carried out;
- Sampling Manual—approaches and protocols for environmental and biological sampling for collection of data compatible with the IEUBK Model;
- Technical Memoranda—occasional technical updates that will be released to explain some features in greater detail or to alert the user to possible misapplications of the model.

1.3 GETTING READY TO USE THE MODEL

1.3.1 Preparing a Site-Specific Exposure Scenario

The use of the IEUBK model requires input data that are appropriate to the site(s) and subject(s). The most convenient way to do this is to construct a multi-media, site-specific exposure scenario using the exposure scenario worksheet (Figure 2-11; see Section 1.4.3).

For most assessments of lead-contaminated soils, the minimal site-specific data are the soil lead and indoor dust lead concentrations for the residential exposure unit. Additionally, it would be helpful to include estimates of specific exposures from diet, drinking water, air, maternal exposure, or other sources that could replace the default exposure parameters believed to be of concern at the particular site.

There may be potentially important differences among sites, and predictions of blood lead values are expected to become more accurate as more site-specific data are added. Children at highest risk are those with the highest exposures to some lead-containing medium. Data should be collected at a site so as to identify locations in the residence or community where young children may be exposed to elevated levels of lead in soil, dust, water, or air. Household-level data are useful because proposed soil, dust, and paint abatements are usually based on the house and yard as the most likely sources of lead exposure in preschool children. High exposures from lead in the household water distribution system are also possible, and this source has been identified in some childhood lead poisoning cases (Cosgrove et al., 1989). The preferred level of environmental input data for the model can be derived from a comprehensive multimedia household environmental lead study.

The households studied should be representative of housing or sites where young children currently reside, as well as the places where young children may live in the future. In many applications, you will also need to include existing homes not occupied by children. These can usually be addressed in the same manner as housing currently occupied by children, using specific measurements of various environmental media lead concentrations. Risk assessments addressing as yet unbuilt housing should use existing residential site soil concentration data.

Predictions of blood lead concentrations may improve with better information on lead concentrations where the child spends time during the day, or on child-specific behavior.

Activity pattern analysis, based on data taken from questionnaires and family interviews, can be useful in identifying children currently at risk, and in determining site-specific differences in behavior or access to lead sources that may differ in bioavailability. Public education and parental awareness of lead hazards may reduce the amount of lead in soil and dust ingested by the child, and quantitative studies of the effects of such actions are currently in progress.

Exposure of children in day care centers, playgrounds or open areas may substantially affect total exposure to lead when potential lead exposures in such areas are high. These cases need to be considered in site risk assessment. The IEUBK Model allows dust and drinking water ingestion components to be separated into household and non-household sources by allocating a percentage of dust and water intake outside the home to sources with other concentrations. Time-weighted average air lead exposures are believed to be adequate indices of lead intake by inhalation in home and non-home settings under most circumstances and are used in the model. However, there is presently little information on the use of time-weighted averages for ingestion of soil, dust, or water away from the home. Soil and dust ingestion depends on children's activities, on hand-to-mouth behavior, and on intensity of soil contact related to sources and pathways away from home.

In addition to exposure, the IEUBK Model also allows site-specific information on the bioavailability of lead from various sources to be taken into account. Bioavailability describes the relationship between the potentially available lead intake from environmental media and the amount of lead entering the body through the lungs or the gut and then into systemic circulation.

You should be alert to the possibility that there may be site-specific differences in bioavailability of lead at different sites, particularly with respect to soil and paint. Some factors that may affect bioavailability include chemical speciation of lead in soil or paint, size of particles, mineral matrix of the particles, and whether the particles are likely to be ingested by the child along with meals or on an empty stomach. These are discussed in Section 4.1. Many of the issues are subtle and should be referred to the EPA Technical Review Workgroup for Lead.

In some cases, relatively non-available environmental lead in soil or paint can be converted into readily available lead particles in household dust by physical and chemical processes in the environment. A housing unit with lead in paint or soil will continue to generate household dust lead exposure as long as paint deteriorates or is disturbed by

remodeling, and as long as outside soil and surface dust are moved into the house by pets and by human activities like gardening and remodeling.

The model default value for the Geometric Standard Deviation (GSD) (reflecting variability among individuals who have contact with a fixed lead concentration) is based on analyses of data from neighborhoods having paired sets of environmental concentration and blood lead data. The recommended default GSD of 1.60 is believed to be very widely applicable. Only when reliable site-specific paired data from a sufficiently large study are available, should the substitution of a site-specific GSD be made using guidance given in Section 4.2.

1.3.2 Understanding How the Biokinetic Component of the Model Works

The general term "biokinetic" is used to describe the movement of lead through various parts of the human body as a kinetic process. Current blood lead concentrations depend on prior exposure history as well as present exposure. With constant lead exposure, a near steady-state blood lead concentration level is achieved because there is a dynamic near-equilibrium between lead moving out (from blood plasma to peripheral tissues and through excretory routes), and lead moving in (to plasma from gastrointestinal uptake and remobilization into plasma from peripheral tissues and long-term bone storage).

The IEUBK Model assumes that skeletal lead turnover occurs relatively more rapidly in children than in adults. The lead in a child's blood is thus a mixture of lead taken up from recent environmental exposure and lead released from skeletal stores that reflect historical exposures. However, the faster turnover time assumed for children compared to adults implies that the lead burden in the skeleton is a smaller fraction of total body burden in children than in adults. The skeletal contribution to blood lead thus increases as the skeletal fraction of total body burden of lead increases.

The blood lead concentrations in children achieve nearly a steady state relationship with exposure within a period of months after changes in exposure. The situation in children is more complicated than in adults because the kinetic parameters also change with the child's growth and with changes in behavior that affect lead intake, absorption, distribution, and elimination. The model is adequate to estimate childhood blood lead concentrations in near-equilibrium or in slowly changing exposure settings, as may be attained some time (months) after abatement occurs. The gradual phase down of lead in gasoline would be an example of

changes that occurred slowly enough in most urban areas to permit accurate modeling of blood lead concentration changes accompanying the air lead concentration changes.

1.3.3 Understanding Limitations of the Model

The IEUBK Model is designed to evaluate relatively stable exposure situations, rather than rapidly varying exposures. The model does not report each iterative calculation; rather, it reports one-year average blood lead concentrations. Because the IEUBK Model allows changes in exposure to environmental lead concentrations only at one year intervals, and provides output at only one year age intervals, changes in exposure are smoothed over one year. The model cannot be used to predict the effects of short term exposure episodes, such as exposure over a few days or weeks to lead dust and airborne particles that may be generated during lead paint abatement. The IEUBK Model should provide reasonable accuracy for blood lead concentration prediction as long as the changes in these environmental lead concentrations can be approximated by annual average values.

The model is intended to describe a single residential-level exposure setting. The dwelling unit could be a detached single-family home, a separate home in a multiple-unit building such as a row house or duplex, or an apartment in a multiple-unit building. There is an implicit assumption that the input parameters characterize long-term residential exposure scenarios in such settings. While exposure changes daily in response to changes in the child's diet and activity, there is presumably a true mean exposure level that can, in principle, be estimated from real-life samples. For this reason, the IEUBK model allows changes in air, food, dust, and soil lead exposure input parameters only at 1-year intervals. Although water lead exposure could, in principle, be handled in similar detail, the IEUBK model does not allow annual changes in drinking water lead during the model run. The IEUBK model includes some capabilities for dealing with lead exposures outside the home, such as by use of separate dust ingestion parameters and concentrations at day care centers, schools, and secondary residences.

We recommend using a simple average or arithmetic mean of soil lead concentrations from a representative area in the child's yard, and an average of dust lead concentrations from representative areas frequented by children inside the house. This rationale is appropriate for areas that are sufficiently small so that any part of the area may be accessible to a typical child living at a random residence located within the area.

The IEUBK model calculates blood lead and tissue lead burdens for all ages from 0 to 84 months. However, the blood lead concentrations in children less than 6 months of age will still be affected by pre-natal lead exposure and are likely to show little influence from exposure to soil, dust, and paint, which are the media currently of greatest interest. The results of the model simulation are therefore not reported for children younger than 6 months.

There are many reasons why individual blood lead concentrations may differ from the predicted geometric mean blood even though the predicted mean accurately describes the population. Some of the components of individual differences are discussed in Section 4.2. The GSD is the only parameter in the model that characterizes the combined variability in blood lead attributable to inter-individual differences and "random" temporal variability in absorption and biokinetics, "random" behavioral changes and inter-individual differences affecting ingestion rate, and measurement errors in environmental lead concentration. The strength of this approach is that GSD estimates are based on empirical data on the variability of blood lead levels in children exposed to similar concentrations of lead. Other approaches to evaluating the effects of variability, such as Monte Carlo simulation, were deferred for the present version of the IEUBK Model, because they demanded excessive computation and require much greater amounts of model input data. Monte Carlo methods, however, are still being evaluated as a possible enhancement of the IEUBK model, as discussed in Section 1.5.

1.4 RUNNING THE MODEL

1.4.1 Your Responsibilities

The IEUBK model provides a great deal of flexibility in describing site-specific or age-dependent exposure scenarios. The price for this level of flexibility is that no exposure scenario is appropriate for every application of the IEUBK model, and this is particularly true of the "default" parameters. The responsible use of the IEUBK model requires input data that are appropriate to the site(s) and subject(s). The most convenient way to do this is to use the exposure scenario worksheet (Figure 2-11; see Section 1.4.3).

The most sensitive parameters for most applications involving soil lead exposure are the soil-to-indoor dust transfer coefficient, the soil and dust ingestion parameters, the soil lead absorption fraction, and the Geometric Standard Deviation. You should always review these parameters.

Factors affecting transport of soil lead into household dust should be noted when appropriate. For example, houses with very small grass-covered yards are likely to have a smaller contribution of the yard's soil lead concentration to household dust lead concentration than houses with large yards, no grass cover, and fine uncompacted surface soils that are easily blown or carried into the house by humans and outdoor pets. While the concentration of lead in exterior dust derived from the soil may be a useful measure of exposure, these data are not usually available because exterior surface dust samples are not usually collected. You are always responsible for the decision to use default values in place of either measured dust lead concentrations or dust lead concentrations estimated from soil lead concentrations.

The proportion of intake in the form of soil vs. dust should be considered carefully, as there may be differences in the bioavailability of lead in soil vs. lead in house dust even when much of the dust is derived from soil. In spite of considerable efforts to determine the ingestion intake of soil and dust by children, these values are still subject to uncertainty. Site-specific data on soil ingestion by children are rarely available, but would be valuable in modeling site-specific exposure to lead. Only limited information is available about the effects of the child's micro-environment on soil and dust ingestion, with evidence suggesting much larger intakes of soil for children in intrinsically dirty environments such as campgrounds, and lower soil intake for children who spend much of their time in cleaner environments such as day care centers.

You are responsible for the choice of non-default bioavailability parameters. Bioavailability parameters may differ among sites. Non-default bioavailability parameters may be justified by experimental studies with the actual site materials, assessments of other sites with similar materials, or site specific information on properties of particles that may affect bioavailability.

The Geometric Standard Deviation is not considered a highly site-specific parameter, and should normally be kept at its default value of 1.60. If you use some other value, you should document the reasons for this modification, since risk estimates are typically very sensitive to the GSD value used.

1.4.2 Exploring Model Options

The IEUBK model has a large number of options. You are encouraged to explore these options before doing any substantive analyses, because there are often several alternative methods that can be used to obtain model outputs. These options are identified in Chapter 2.

They include alternative source menus for soil and dust lead, dietary lead, and lead in drinking water. The soil/dust lead menu includes options for air-to-dust and soil-to-dust transfer coefficients, as well as for non-household sources.

There are options beyond single runs of the model. These include multiple runs for overlay plotting of probability curves, for plotting blood lead vs. environmental media lead concentration, and for multiple runs (batch mode input) for each of a group of individual children of different ages using child-specific data.

The multi-media bioavailability menu includes options for changing the passive vs. facilitated absorption of lead from all media. The half-saturation uptake, a parameter that determines the extent of non-linear or saturable absorption, may also be changed from the normal default value of 100 μg Pb/day.

Run options include the choice of an iteration time step. With low exposure and no year-to-year change in concentration, as used in the "Default" option, there should be no differences in output using other iteration time steps. Differences in blood lead of a few percent may occur with higher and rapidly changing exposures. For a single run, almost any PC (XT or later) will produce a solution within 60 seconds, even without a math coprocessor, with the default iteration time of 4 hours. However, with a batch mode input file of several hundred records, the simulation run may take many hours. In this case, you may select a longer iteration time and speed up the run for a preliminary analysis. If you use a longer time step, you should verify accuracy using records with high exposure or large changes in exposure.

1.4.3 Documentation of Input Parameter and Data Files

By reviewing every adjustable parameter in the model and noting which ones have been modified in a particular run, you have a permanent record of the input. An electronic copy of the exposure input parameters can be made using the parameter SAVE option. Distinctive names for parameter files ([name].SV3), input data files ([name].DAT), simulation run files (RESULTS.TXT), batch mode output files ([name].TXT and [name].ASC), probability plot overlay files ([name].LAY) and blood lead vs. media concentration files ([name].MED) may be used to document input specifications as well as output.

The worksheet provides a convenient format for noting reasons for use of non-default parameters, or justification for use of default parameters. For example, soil lead

concentrations and dust lead concentrations could be measured values at each house. Repeated values of household data would be used to weight the statistical results from batch mode files. Missing value imputation methods should be identified, for example, "KID ID = 17,22,35, missing dust lead concentration estimated by PbD = 180 + 0.28 * PbS." This is critical information in allowing other users to reproduce your results (including yourself, since it is unlikely that most users will be able to recall over one hundred model parameters after the passage of some months or years).

1.4.4 Documentation of Model Output

1.4.4.1 Selecting Output Alternatives

Results of IEUBK model simulations may be saved in several forms. You should select in advance the most useful of these forms, since the results of some interactive simulations cannot be recovered once you have bypassed the opportunity to save the results. Choices are:

(1) A sequence of single simulation runs. Sequential runs can be interactively appended to the file named RESULTS.TXT. The average of the geometric mean blood lead concentrations for children in sequential one-year age intervals, the input concentrations for several media, and the media-specific daily lead uptake for each year are saved. You must use the "Save" option at the end of each run to be saved, but this allows you to drop results from non-informative runs rather than save them.

(2) A sequence of graphics overlay simulation runs. The multiple plot option saves input data for blood lead probability plots for a range of evenly spaced media lead concentrations. For example, you may generate plot data for soil lead concentrations of 250, 500, 750, and 1000 ug/g, for children of ages 12 to 24 months. The data in the [name].LAY overlay file includes the geometric mean blood lead for children in the age range, the lead concentration in soil and in other media. The actual plots of probability density or cumulative distribution functions depend on the GSD value selected, and these plots include the probability of exceeding the user-specified LOC for use in risk estimation. Probability plots may be printed on standard laser printers.

(3) A sequence of blood lead vs. media lead simulation runs. The media range option saves input data for blood lead vs. media lead plots for a range of evenly spaced media lead concentrations. For example, you may generate plots of blood lead vs. soil lead concentrations smoothly interpolated from calculated values at 250, 500, 750, and 1000 ug/g, for children of ages 12 to 24 months. The data in the [name].MED overlay

file includes the geometric mean blood lead for children in the age range at the selected media lead concentrations, the lead concentration in soil and in other media. Plots may be printed on standard laser printers.

(4) Batch mode simulation runs. The batch mode option requires an input data file, as described in Chapter 2. Output consists of user-named files [name].ASC and [name].TXT that contain predicted blood lead concentrations for each case or record (child) in the input data file. The output files also document the missing value imputations when some of the input data on residential lead concentrations in air, water, soil, or dust are missing. The files may be used as input for the statistical analysis programs in the companion PBSTAT program, which produce statistical and graphical comparisons of the observed and predicted blood lead concentrations.

1.4.4.2 Understanding the Output

You should carefully review the output options described in Section 1.4.4.1. Each option allows you to examine a different aspect of the IEUBK simulation. The numerical simulation component of the IEUBK model produces an estimate of a geometric mean blood lead concentration for children of a given yearly age. This is the average of the estimates for children during that one-year interval. The IEUBK model arrives at these estimates by calculating at each time step an updated estimate of all compartment lead masses, or equivalent tissue lead concentrations. The update algorithm combines uptake of lead from the environment with all of the movements of lead into each compartment from another compartment, or out of each compartment, either into another compartment or by elimination from the child's body. In this version of the IEUBK model, the output consists of the daily uptake rate (intake rate times fraction absorbed) for each medium, and the blood lead concentration, as annual averages.

The output from a single simulation run may be displayed in several forms. Most users wish to see the variability associated with a predicted blood lead concentration. This range can be demonstrated graphically by selecting the intrinsic variability GSD and then plotting a cumulative probability distribution. The range of plausible blood lead values may be determined graphically as defined by upper and lower percentiles of the distribution. For example, the 5th and 95th percentiles of the distribution will include 90 percent of the children with the given site-specific or household-specific exposure scenario. Since "plausible range" requires a subjective choice of percentiles, you are free to choose any appropriate values. Since the predicted geometric mean blood lead concentration is based on

an a priori mathematical simulation and not on a data-driven statistical estimate, this plausible range sh :ld never be considered as equivalent to a confidence interval.

The other output characteristic that many users wish to see is the estimated probability of exceeding the specified blood lead level of concern, corresponding to the given exposure scenario or scenarios (for multiple runs in a given medium). This also requires a GSD value. This probability may be interpreted as the percentage of children with the given household-specific exposure scenario who are expected to exceed the level of concern. If applied to a single site or residence, it may also be interpreted as the probability of exceeding the level of concern for any single child who may reside at that site in the future.

1.4.4.3 Interpreting the Output and Communicating the Results

The model calculates the probability that a blood lead concentration derived from the model's specified parameters will exceed a level of concern specified by the user. There are two valid interpretations for the output:

(1) The output of the model may be considered to be the best estimate of a plausible range of blood lead concentrations for a hypothetical child with a specific lead exposure scenario. The range of values is centered on the geometric mean blood lead concentration expected for a typical child with this exposure scenario. The upper tail of the probability distribution provides an estimate of the risk of exceeding some blood lead level of concern for a typical child of that age residing in the same household and with the same exposure history.

(2) The output of the model may also be considered to be the predicted geometric mean blood lead of a *population* of children with the same lead exposure scenario, and the upper tail of the probability distribution to be the fraction of children exceeding the chosen blood lead level of concern when all of these children have the same exposure history.

The array of applications for which the IEUBK model can be validly used is:

A: One location
- A1: one living unit, one child;
- A2: one living unit, more than one child;
- A3: more than one living unit, more than one child, homogeneous media concentrations;

B: Multiple locations, one neighborhood, homogeneous media concentrations

C: Multiple locations, one neighborhood, heterogeneous media concentrations;

D: Multiple locations, more than one neighborhood, heterogeneous media concentrations;

A single run of the IEUBK model is sufficient for categories A and B. A classification or disaggregation of the neighborhood into distinct exposure subgroups is required in categories C and D, with the possibility of different ingestion or absorption parameters for different neighborhoods in category D. Neighborhood-scale and community-scale risk estimation requires aggregating the risk estimates for individuals or subgroups.

The differences between these levels is sketched in Figure 1-3. Category A requires calculating only a single blood distribution. Category B requires calculating a blood lead distribution for each child, but since each child of the same age has the same exposure scenario in category B, a single run of the model is sufficient to characterize risk for this subgroup. In category C, there are different exposure scenarios for each subgroup. Risk estimates must be calculated for each such subgroup, then added up across sites and children.

The model output in category A: Single child, single site of exposure, includes a blood lead concentration, a distribution of blood lead concentrations, and a probability of exceeding the blood lead level of concern. Since children in environments with the same lead exposure may have a range of blood lead concentrations, we describe the likely variability in blood lead for a child with a given exposure scenario by a probability distribution. The predicted blood lead concentration is the geometric mean of the distribution of blood lead concentrations that may occur for a typical child with the specified exposure scenario. Risk is calculated from this distribution as the probability that a hypothetical child living at this site, with the specified exposure scenario, will have a blood lead concentration exceeding the blood lead level of concern. This single-child assessment is necessary in order to use the model to evaluate remediation options on a house-by-house or yard-by-yard basis. The single-child assessment also provides a criterion for model testing and validation using epidemiology data.

The model output in category B: Multiple children, single site or equivalent sites of exposure, is the predicted blood lead concentration for each child as the geometric mean of the distribution of blood lead concentrations that may occur for each child with the specified exposure scenario. Risk is calculated by aggregating the calculated risk for each child as the percentage of hypothetical children living at this site or at these sites, with the specified exposure scenario, that will have a blood lead concentration exceeding the blood lead level of

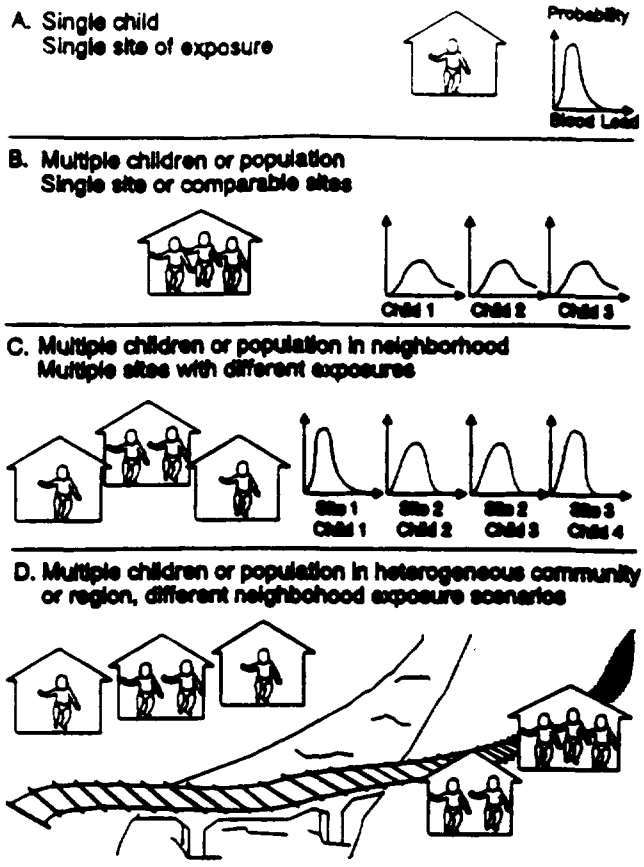


Figure 1-3. Categories of application of the IEUBK Model.

concern. The calculation is exactly the same as the single-child assessment, but there is an important shift in interpretation of the output.

There are situations in which a single site really can have multiple children of the same age with the same exposure scenario. A single housing unit may be occupied by several households with pre-school children of the same age. Rental properties may be occupied in succeeding years by different families, each of which may have a pre-school child of the same age with virtually the same exposure as occupants in other years. In general, the multiple-child or population exposure scenarios would be applied to a hypothetical population of occupants.

Neighborhood-scale risk estimation is discussed in Section 4.2, with examples. The model output in C: Multiple children, multiple sites with different exposure, cannot be

obtained by a single run of the IEUBK model. It is necessary to construct an exposure scenario for each distinct exposure subgroup in the population. For each child or exposure subgroup, risk is calculated in a single run of the IEUBK model with the specified exposure scenario. The risks for each exposure subgroup are aggregated across all subgroups, weighted by the number of children with that exposure scenario or by the percentage or likelihood of the exposure scenario.

There is no one-step method by which neighborhood-scale risk estimation can be done using this version of the IEUBK model. The problem of risk estimation for children in a large community or a region is even more difficult when different subgroups of children may have very different exposure scenarios, including differences in behavior that affect ingestion, and differences in lead absorption due to behavioral or nutritional differences.

A common misinterpretation of the IEUBK Model is that it predicts *community* geometric mean blood lead and the fraction of children at risk when the input is the mean or geometric mean of household-specific environmental lead concentrations. That mis-step can be misleading, particularly when the environmental variables have a wide distribution among the neighborhoods of the community. This misinterpretation is especially dangerous for post-abatement settings intended to eliminate the higher exposures when there are multiple exposure media. A correct approach requires applying the model to each individual home or site using the lead concentrations seen at that site and combining these results as an aggregate of sites in several neighborhoods to form an estimate of community risk. A second useful approach is based on subdividing a community into neighborhoods and clusters of residence units with similar media lead concentrations. Specific information on building appropriate neighborhood exposure scenarios is given in Section 2.3, *Building an Exposure Scenario*. Examples are provided in Section 4.2.

We should emphasize that the IEUBK model is intended to provide a best estimate of geometric mean blood lead. The IEUBK model is not intended to be used in a worst-case scenario, as the model does not apply any uncertainty factors or modifying factors in making risk estimates. If, as usual, there is some uncertainty about model parameters, these can be evaluated using sensitivity analyses. Remember that you are responsible for documenting plausible non-default values.

Uncertainty about parameters is not the same as the intrinsic variability in environmental data and blood lead responses. The components of variability are discussed in

Section 4.2 on the blood lead Geometric Standard Deviation (GSD), which plays a critical role in risk estimates.

1.5 REFINEMENTS AND ENHANCEMENTS

The biokinetic component of the IEUBK model is based on an age-dependent compartmental model with identifiable physiological compartments: red blood cells, plasma and extracellular fluids, kidney, liver, other soft tissues, trabecular and cortical bone (Figure 1-1). There are many compartmental models in the literature; some with fewer compartments (Rabinowitz et al., 1976), others with many more compartments (Leggett, 1993). The Technical Review Workgroup for Lead was aware of important research in the development of physiologically-based pharmacokinetic (PB-PK) models for lead in humans, primates and rats that took into account the slow diffusion of lead through the bone matrix (O'Flaherty, 1992a,b,c, 1993a,b). However, the Workgroup chose to develop a compartmental model that uses transfer times or transfer rates between compartments instead of physiologically based compartmental coefficients. The transfer rates can be estimated from data in non-human primates, especially the studies on infant and juvenile baboons that were done at New York University (Mallon et al., 1983; Harley and Kneip, 1985).

The IEUBK biokinetic model was based on:

(1) empirical kinetic data on blood lead in baboons of similar weight and developmental stage to human infants and young children;

(2) kidney, liver, tibia and femur lead concentrations in baboons after the end of the lead exposure study;

(3) autopsy data for lead levels in young children who died from causes not related to lead exposure;

(4) extrapolations from studies in human adults;

(5) lead feeding and lead balance studies in human infants.

There is, in principle, a degree of similarity between these approaches, since the compartments in the IEUBK model are defined by real anatomical and physiological properties. The transfer times from the PB-PK model can be calculated from blood flow rates to organs and tissue groups, volumes of these organs, partition coefficients across

membranes, and solid state diffusion coefficients for the bone matrix. The principal difference between the biokinetic components of the IEUBK and PB-PK models is that, in the absence of suitable physiological data, empirical data were used in estimating transfer times in the IEUBK model. Future development of the IEUBK Model is expected to continue in the direction of physiologically based biokinetic components similar to PB-PK models.

Many users have expressed interest in tools that allow a more detailed investigation of the effects of non-environmental variability on the distribution of blood lead concentration. The Monte Carlo approach would allow every parameter in the model to be assigned a random variation at every iteration of the computation. For example, each parameter could be multiplied by a random factor (mean value 1) at every iteration. This would require that adequate data would be available to support the input distributions. An extremely large amount of computing would be necessary. A substantial amount of additional study is needed before Monte Carlo methods can be added to the IEUBK model.

The IEUBK model currently evaluates children from birth to age 84 months. Many users have requested extension of the model to other populations, including older children and adults, with emphasis on populations at special risk. Both the physiological and biokinetic parameters of adults are at least as well known as those of children, with the possible exception of lead distribution within the human maternal-fetal unit. Transfer of lead from the mother to the neonate during lactation would also be of interest.

1.6 GETTING MORE HELP

As scientific knowledge advances, this Guidance Manual will be updated and revised. If you have questions regarding the site-specific application of the IEUBK Model, you may direct your inquiries to the appropriate EPA Regional Toxics Integration Coordinator. Comments on the technical content of the manual or suggestions for its improvement may be brought to the attention of members of the EPA Technical Review Workgroup for Lead ··ed in the front of this document.

2. A GUIDED TOUR THROUGH THE LEAD MODEL

2.1 THE LEAD MODEL IS DRIVEN BY MENUS

Environmental Protection Agency's Integrated Exposure, Uptake, and Biokinetic Model for Lead in Children (IEUBK Model) is a microcomputer program that performs many different functions related to estimating blood lead levels in children. The overall model functions are sketched in Figure 2-1.

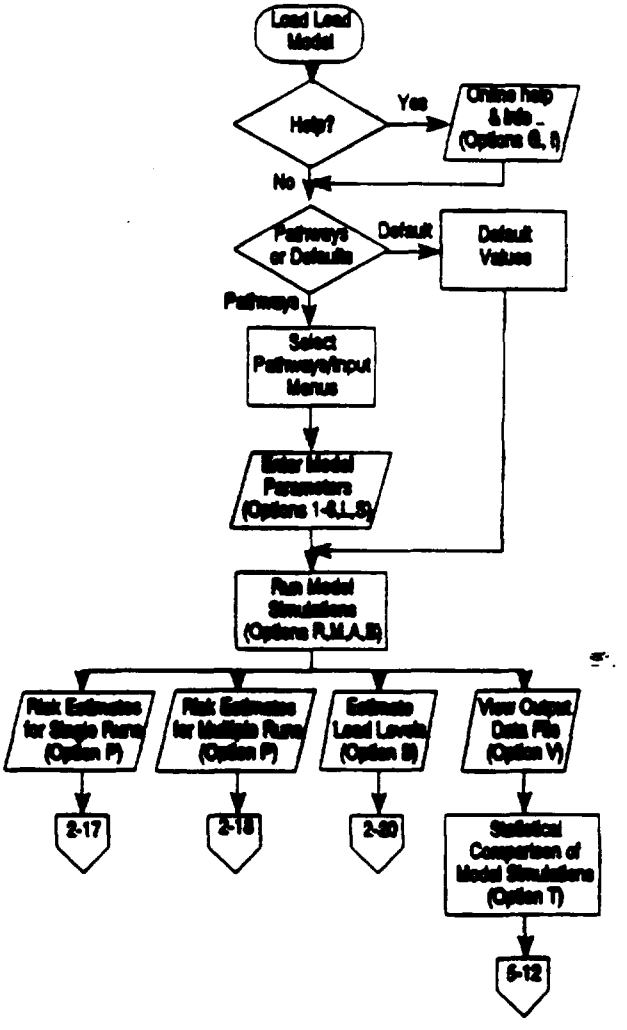


Figure 2-1. Schematic diagram of the overall functions of the lead model. Numbers in pentagons indicate sections in this document containing more detailed information.

The oval shapes are terminal steps (i.e., the beginning or end of a function or option). Rectangles show internal processes and rhomboids show user data entry operations or functions. Diamond-shaped figures are decision points where the user must choose one of the model options on a list. The "NO" branch usually follows the model's baseline or "default" parameters and functions. Horizontal and vertical arrows refer the user to another figure or page.

The IEUBK model is menu-driven, with on-line help available in almost any menu. The main menu, where any use of the IEUBK model begins, is shown in Screen 2-1. There are five numbered options:

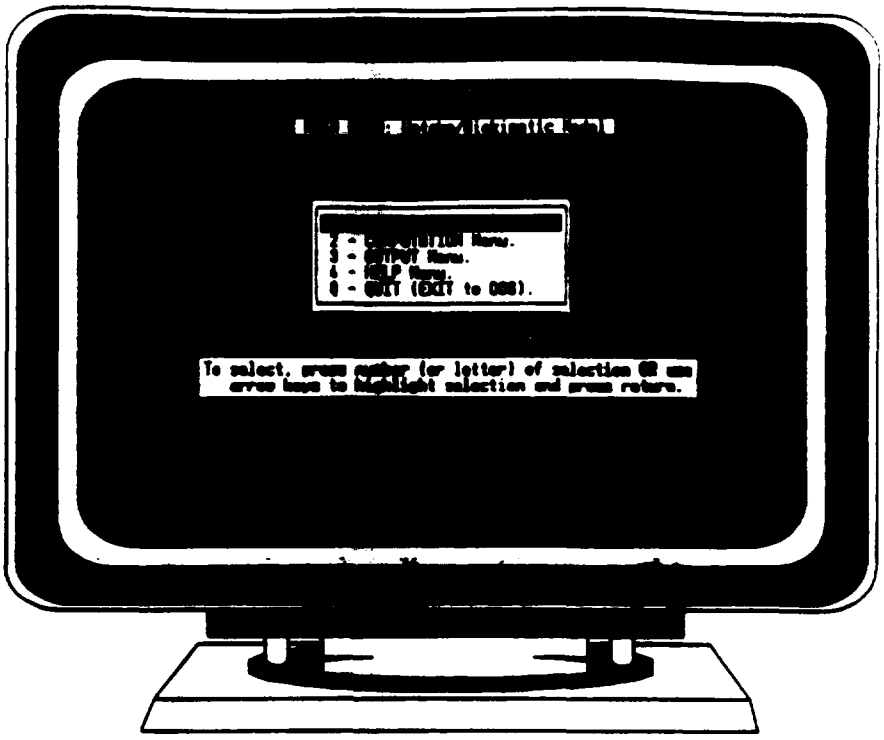
1. Parameter Input Menu
   - 1: Air lead menu
   - 2: Dietary lead menu
   - 3: Drinking water lead menu
   - 4: Soil/Dust lead menu
   - 5: Alternative lead source menu
   - 6: Maternal lead menu
   - L: Load pre-saved parameter input menu
   - R: Return to Main Menu

2. Computation Menu
   - 1: Run a single model simulation
   - 2: Multiple simulation runs with a range of values
   - 3: Blood lead versus media with a range of values
   - 4: Multiple simulation runs with batch input (input data file for each child or household)
   - R: Return to Main Menu

3. Output Processing Menu
   - 1: Save program parameters to file
   - 2: Plot graphs of blood lead distributions
   - R: Return to Main Menu

4. Help Menu
   - 1: General information
   - 2: Information about menus plus help in other menus
   - R: Return to Main Menu

5. Quit
   - Q: Return to DOS prompt.



Screen 2-1. The main menu.

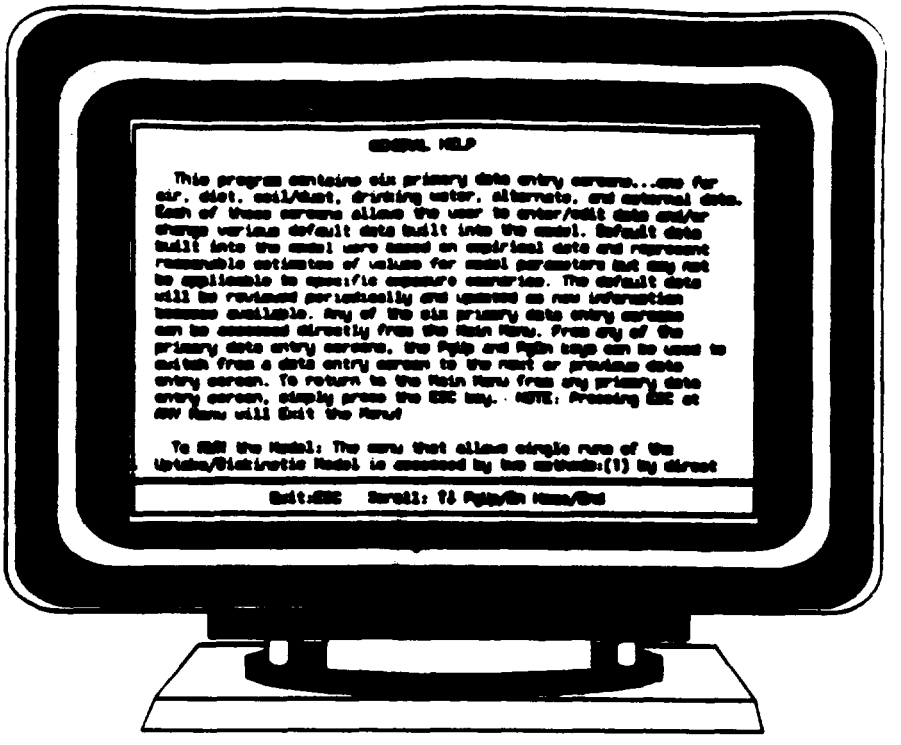
We will briefly discuss the options in each of the input menus. Scientific justifications for the options and guidance values are provided in Section 2.3.

2.2 DETAILED DESCRIPTION OF MENUS

2.2.1 Help Menu (4)

2.2.1.1 General Help (1)

The General Help menu provides on-line information on the data or parameter entry menus, menu selections for running single or multiple model simulations, and use of keyboard keys. This information is shown in Screen 2-2.



Screen 2-2. The general help menu.

2.2.1.2 Information Menu (2)

The Information Menu provides on-line information on the parameter save and load file options, on multiple-run and output processing menus. The information is presented here in Screen 2-3.

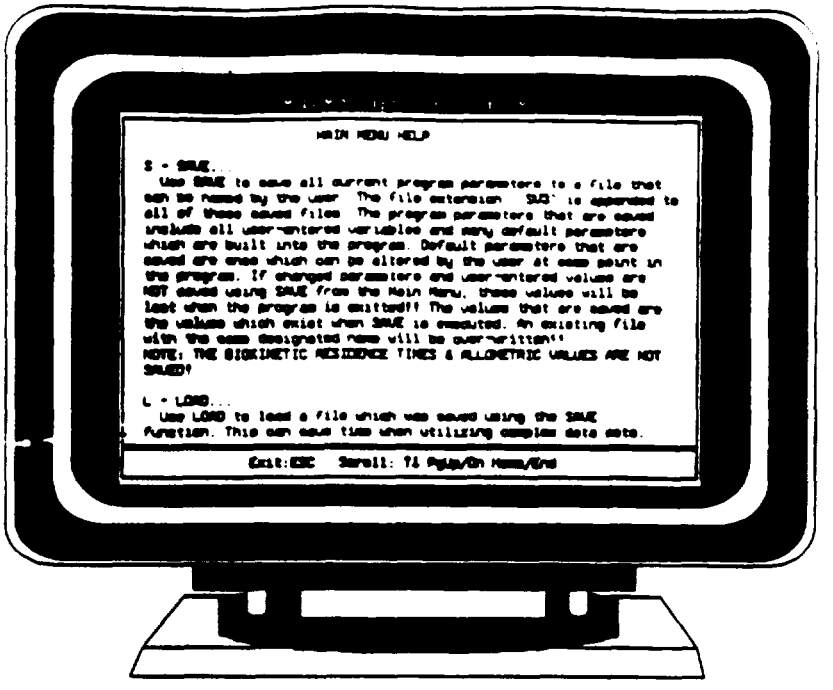
2.2.1.3 Other On-Line Help Menus

Most menu screens contain additional information on the lower part of the screen. Additional information screens are available on specific menu options.

2.2.2 Parameter Input Menus

2.2.2.1 Air Lead (1)

The Air Lead input parameter menu is shown in Screen 2-4 and schematically in Figure 2-2. The air lead concentration is set initially to a typical 1993 urban value of 0.1 $\mu g/m^3$ (U.S. Environmental Protection Agency, 1991c). It is assumed that the indoor air



Screen 2-3. The information menu.

Screen 2-4. The air lead menu.

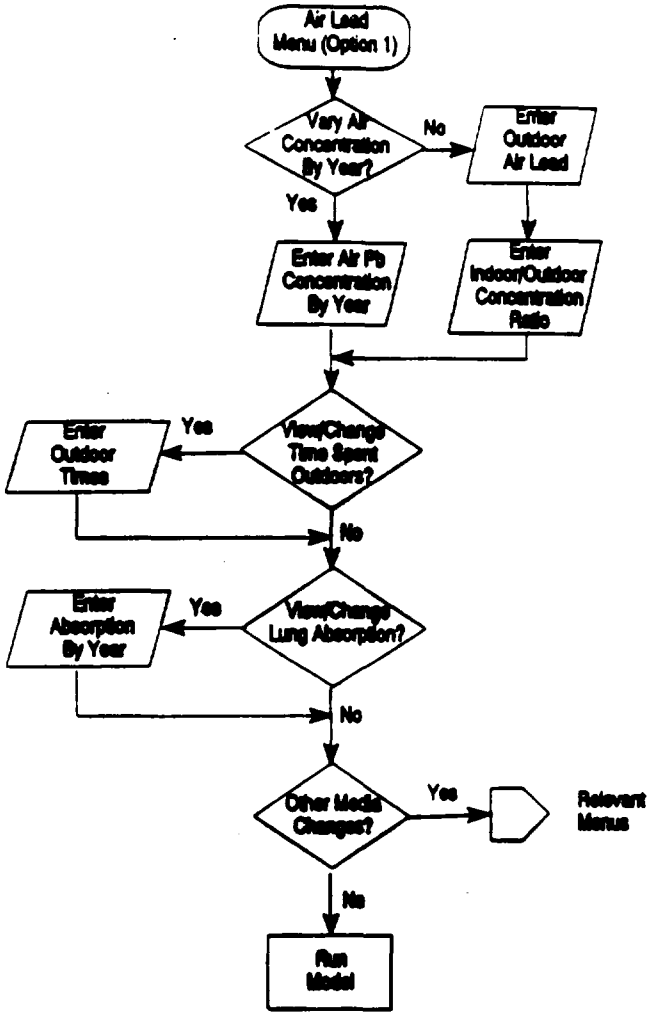
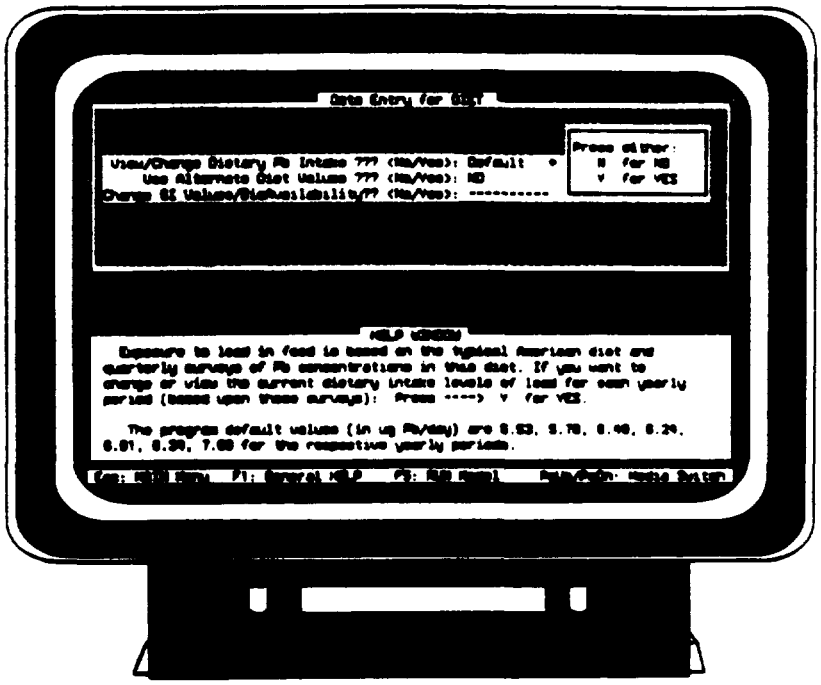


Figure 2-2. Decision diagram for the air lead menu options.

lead concentration is 30% of the outdoor concentration (i.e., 0.03 $\mu g/m^3$) initially. The time spent outdoors and ventilation rate are assumed to depend on the child's age. These parameters allow a time-weighted air lead intake to be calculated; 32% of that intake is absorbed through the lungs into the child's blood. All parameters except the indoor/outdoor air lead concentration ratio may be changed by entering YES in the first line. Some are age-specific values.

2.2.2.2 Dietary Lead (2)

The Dietary Lead input parameter menu is shown in Screen 2-5 and schematically in Figure 2-3. The daily dietary lead intake values for each age apply to a typical U.S. child in a typical setting in the United States after 1990. These dietary lead values may be altered by entering YES to the query "View/Change Dietary Pb Intake?" During the period 1982-1989 there was a distinct reduction in food lead generally attributed to the replacement of lead-soldered cans and the removal of lead from gasoline. Since 1990, food lead in U.S. supermarket food has remained relatively constant. Dietary lead ingestion for years prior to 1990 are given in Section 2.3.2.



Screen 2-5. The dietary lead main menu.

If the dietary lead sources are non-standard, usually because of suspected contamination of fruits, vegetables, fish and meat raised locally or otherwise lead-contaminated, the user can enter specific values by responding YES to the query, "Use Alternate Diet values?" This invokes the alternative menu shown in Screen 2-6.

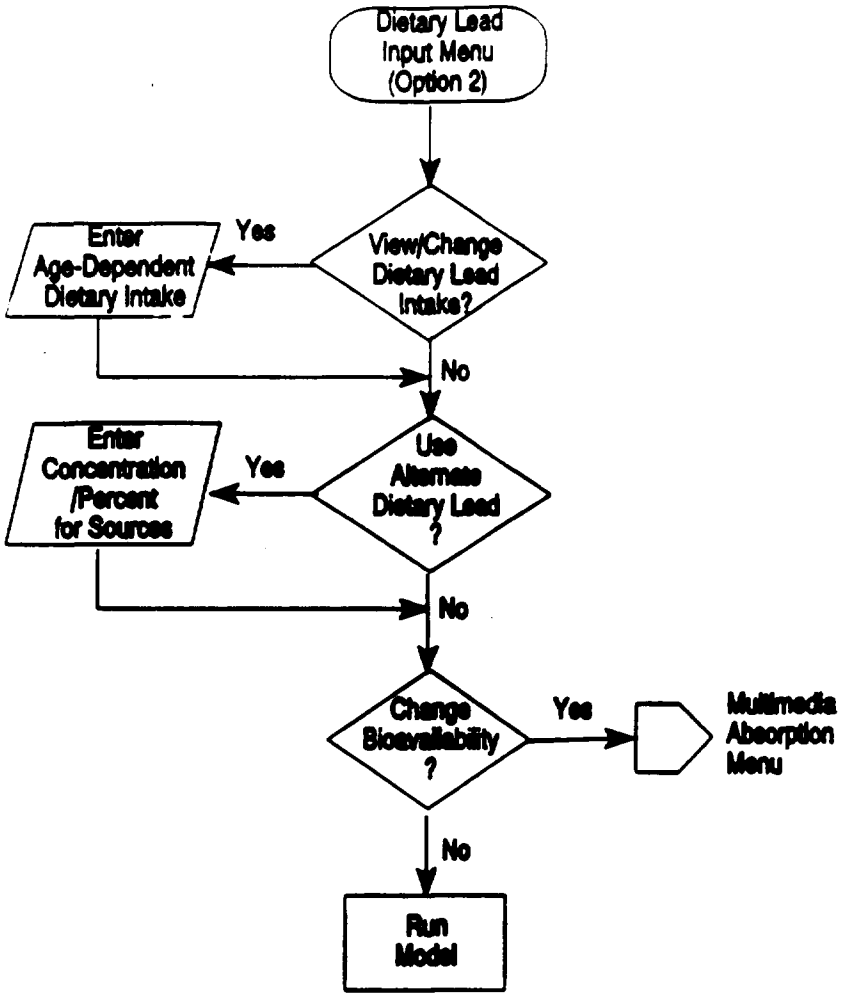
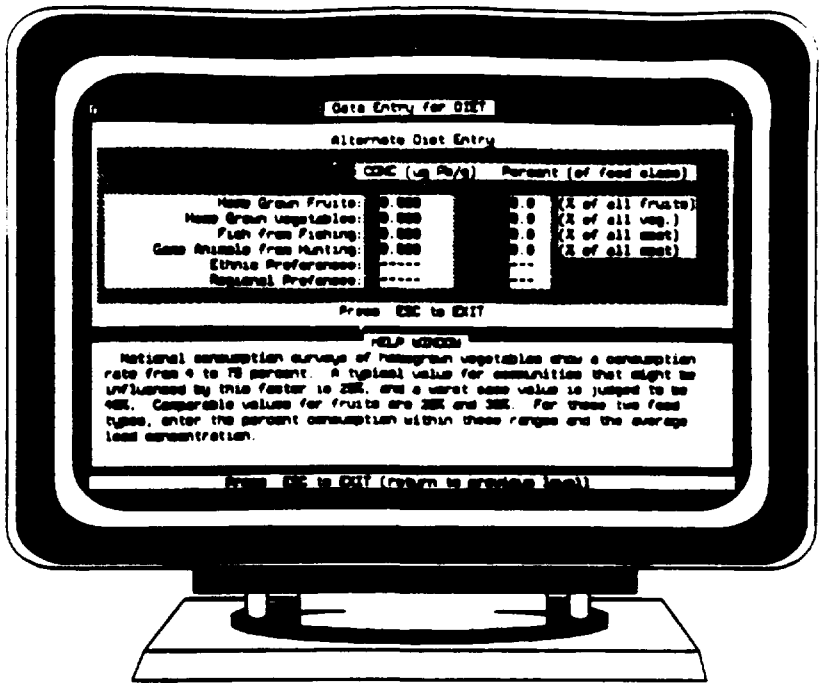


Figure 2-3. Decision diagram for the dietary lead menu options.

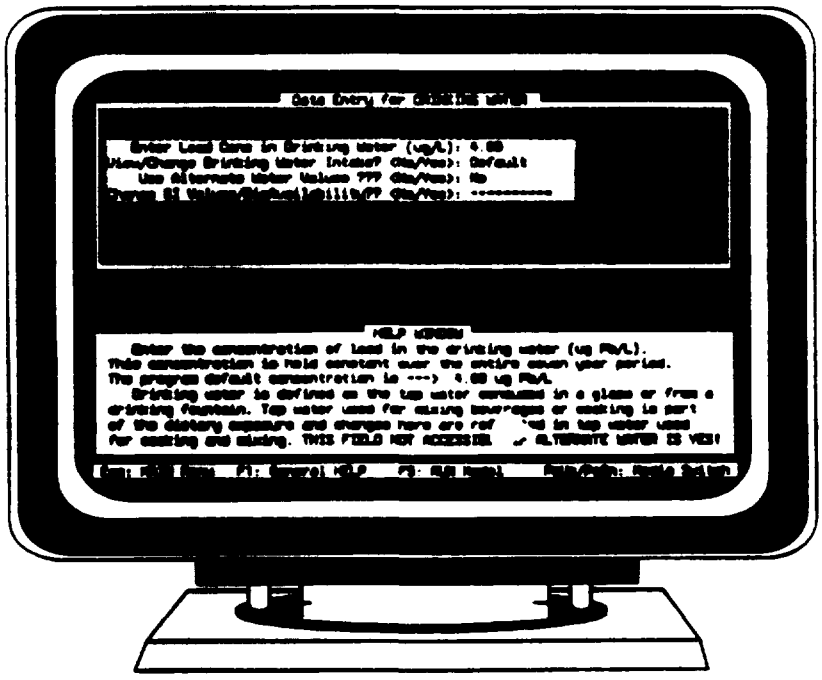
2.2.2.3 Drinking Water Lead (3)

The Water Lead input parameter menu is shown in Screen 2-7 and schematically in Figure 2-4. The water lead concentration is set initially to a typical 1990 urban value of 4 μg/L (Marcus and Holtzman, 1990). The age-specific ingestion of tap water is described in Section 2.3.3.2. Consumption may be modified by responding YES to "View/Change Drinking Water Intake?" and entering new values, as shown on Screen 2-8.

Alternative information may be available in the form of measured lead concentration and percentage of tap water intake from water fountains or other outside sources, and water



Screen 2-6. The alternative dietary source menu.



Screen 2-7. The drinking water lead main menu.

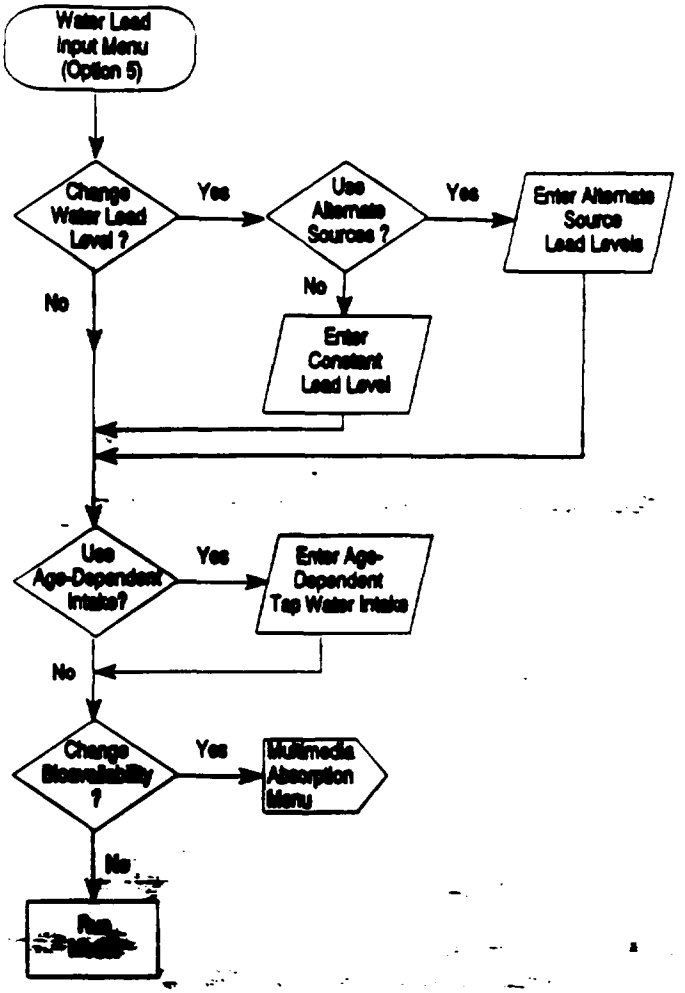
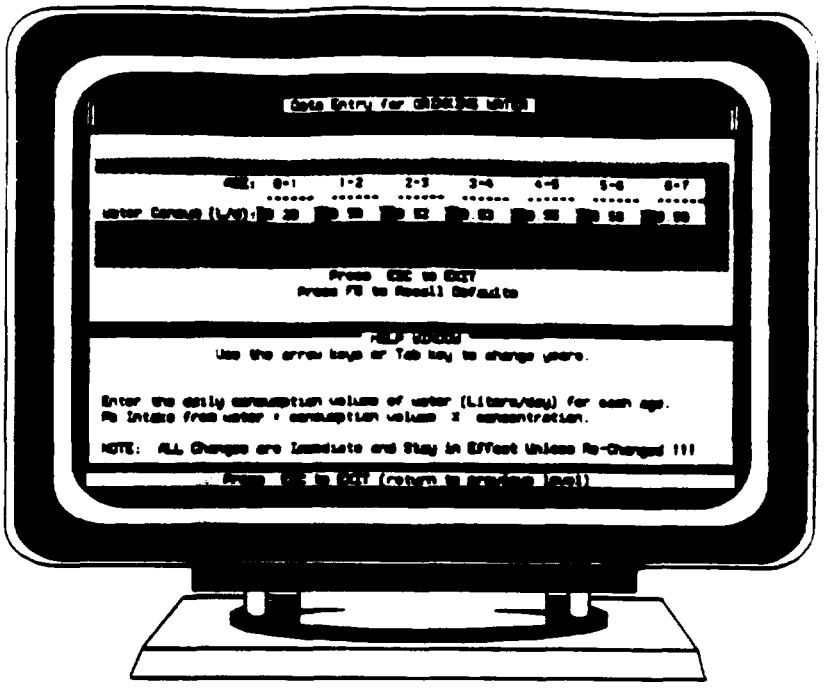


Figure 2-4. Decision diagram for the drinking water lead menu options.

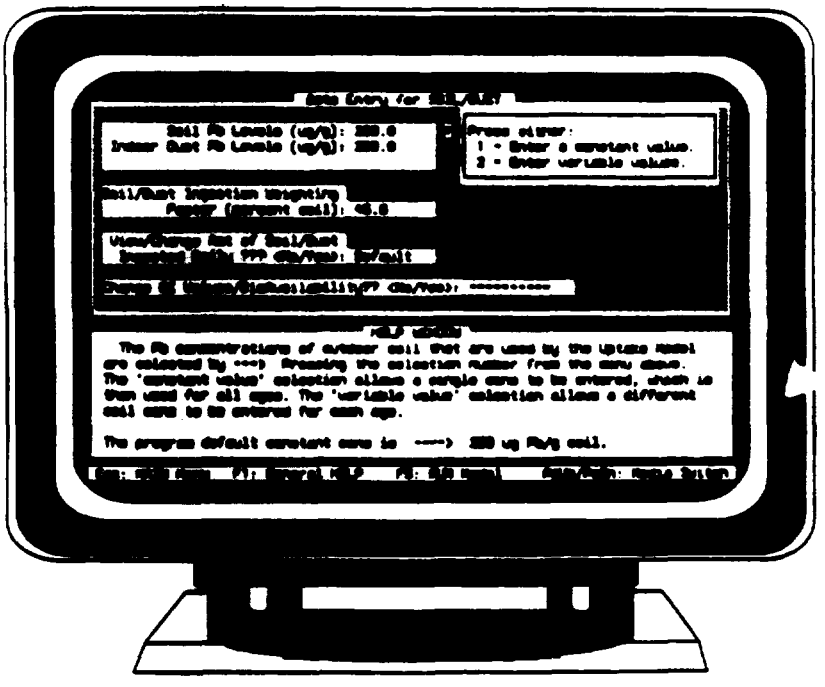
consumed at home in first-draw or flushed modes. This may be entered by responding YES to "Use Alternate Water Values?"

2.2.2.4 Soil and Dust Lead (4)

The Soil and Dust Lead input parameter menu is shown in Screen 2-9 and schematically in Figure 2-5. The soil and dust lead concentrations are set initially to a value of 200 $\mu g/g$. The age-specific ingestion intake of soil and dust combined was estimated from the EPA/OAQPS staff paper on Exposure Assessment Methodology and Validation for the first



Screen 2-8. The age-specific drinking water consumption menu.



Screen 2-9. The soil and dust main menu.

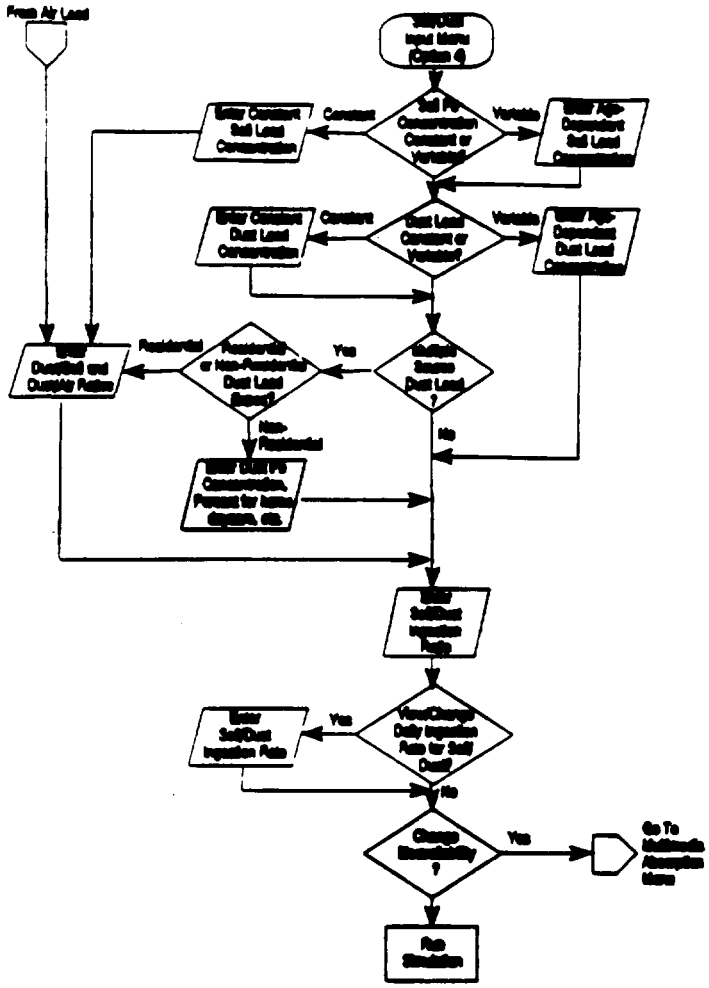


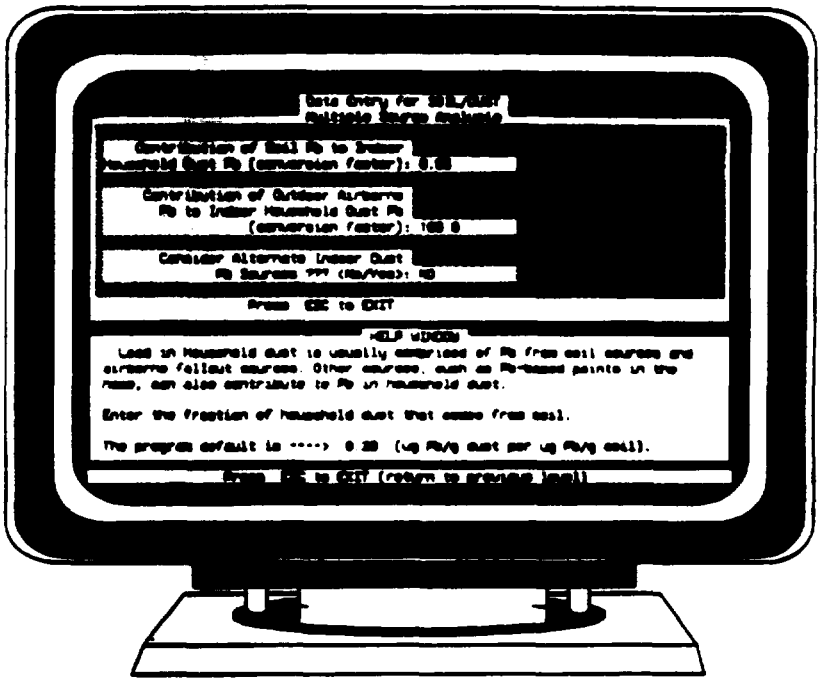
Figure 2-5. Decision diagram for the soil/dust lead menu options.

version of the UBK model (U.S. Environmental Protection Agency, 1989a). Both concentration and intake may be modified by the user.

As shown in Screen 2-9, both soil lead and dust lead concentrations may be changed on a yearly basis by user selection "2", allowing the user to construct reasonable site-specific scenarios.

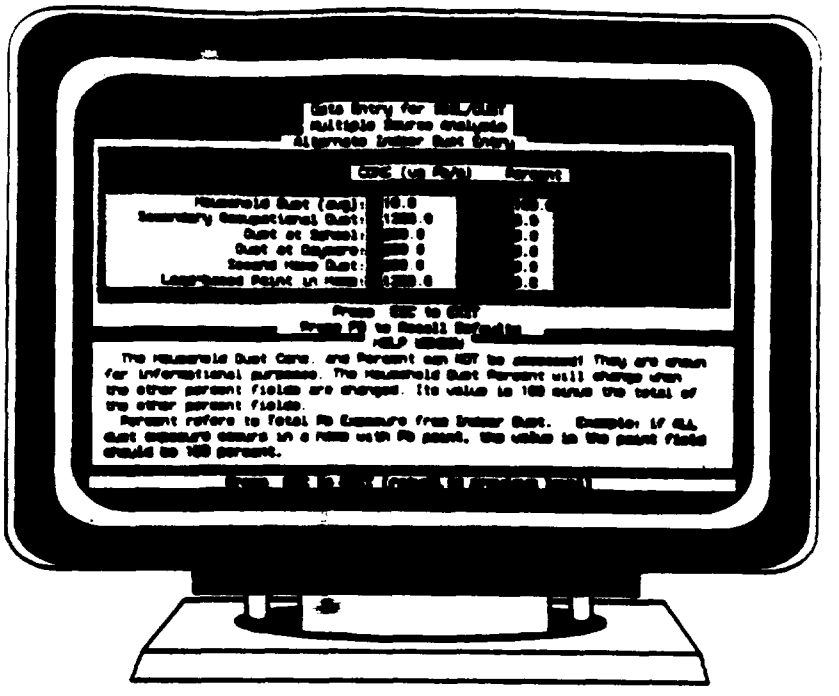
The multiple-source option ("3") on the dust entry line allows the user to use information about the contribution of soil lead, air lead, and other sources to household dust

lead. The Data Entry Screen for the Multiple Source Analysis (Screen 2-10) has three data entry lines. The first line is the *fraction of the soil lead concentration* that contributes to the *concentration of lead in household dust*. If there were no other sources, this would be the ratio of the dust lead concentration to the soil lead concentration. The current default value of 0.70 is appropriate to neighborhoods or residences in which loose particles of surface soil are readily transported into the house. The second data entry line is the contribution to household dust from the deposition of airborne lead, over and above the soil lead contribution. The current default value is an additive increment of 100 $\mu g/g$ lead in house dust for each μg Pb/m^3 air.



Screen 2-10. The multiple dust source menu.

The third line asks whether the user wants to add other sources. If "Yes", then the Multiple Source Analysis Screen is replaced by the Alternative Indoor Dust Entry screen (Screen 2-11). The user may assign both the concentration and percentage of dust intake to baseline household dust, secondary occupational dust, dust at school, daycare, or second home, and the exposure to lead in dust from household paint measured as a percentage of total dust ingestion and its concentration. The default dust lead concentration in the



Screen 2-11. The alternative indoor dust menu.

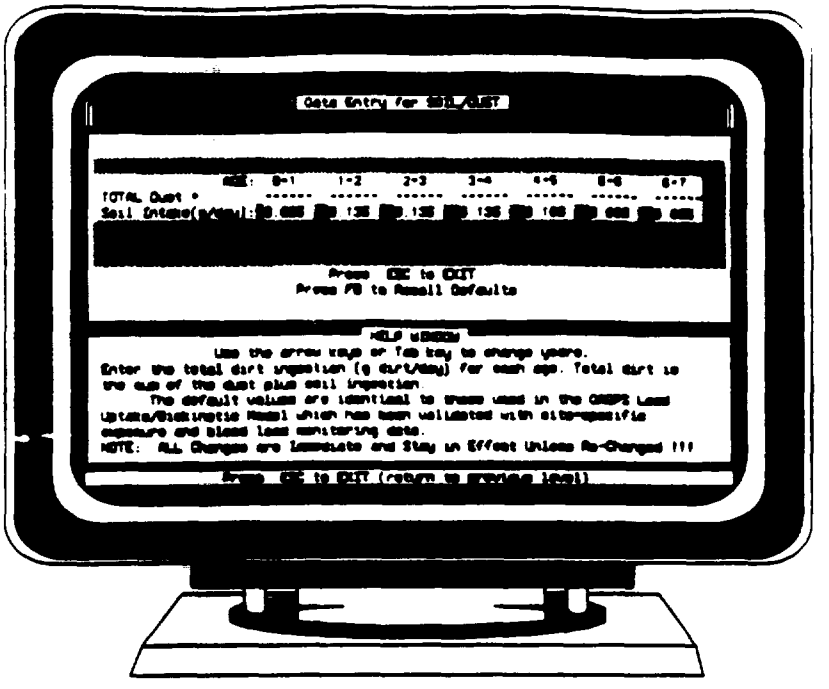
Alternative Indoor Dust Entry screen is 100% of household dust at 150 $\mu g/g$. If the Alternative Source Analysis is not used, then the default dust contribution consists of 70% of the soil concentration plus 100 times the air lead concentration. For default conditions, the total dust lead concentration equals 150 $\mu g/g$.

If non-residential exposures to soil/dust are important, the user may access the multiple non-residential source menu.

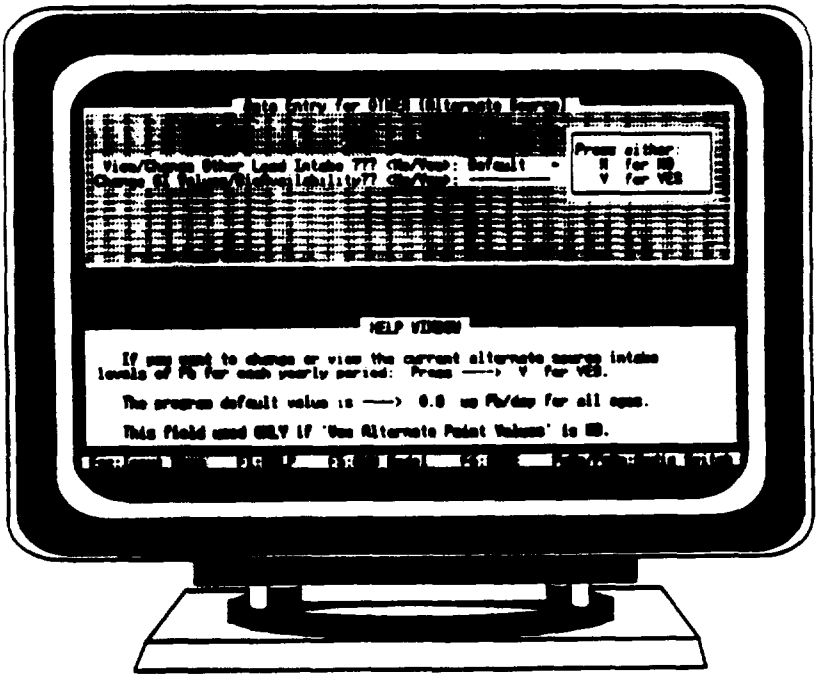
The combined soil/dust ingestion rate (grams total soil + dust per day) can be changed from the current default values in Screen 2-12.

2.2.2.5 Alternate Source (5)

The alternate exposure source menu is shown in Screen 2-13 and schematically in Figure 2-6. The default daily lead intake value for each age is set to 0 μg Pb/day. The user has the option to input any source not otherwise covered by other menus. Examples might be the direct ingestion of lead-based paint, cosmetics or home remedies. In this case, the amount of lead per day needs to be calculated from the information available. If the



Screen 2-12. The soil/dust ingestion rate menu.



Screen 2-13. The alternate lead source menu.

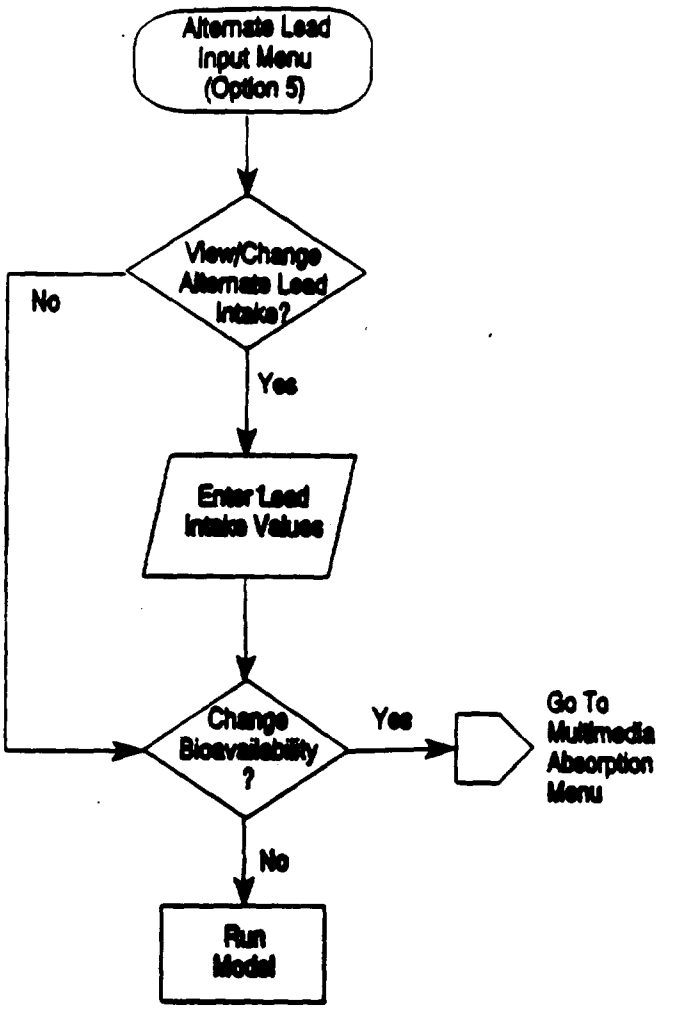


Figure 2-6. Decision diagram for the alternate lead source menu options.

alternative source is lead-based paint (LBP), this exposure would be in addition to exposure to lead-based paint in house dust, which is Option 4 in the multiple source menu of soil and dust. See Section 4.7.1 for a discussion on issues in the use of the model for paint chips.

Building an exposure scenario using this option should be done with care. The model assumes all entries represent chronic exposure. In the example above, the child would require immediate medical attention. Remember that the model output represents only those children defined by the exposure scenario.

2.2.2.6 Bioavailability of Lead in Food, Drinking Water, Soil, and Dust

Bioavailability or absorption of intake from the gut or lung into the blood is a key element in relating external exposure to body burden. Lead intake from media with low bioavailability poses much less of a hazard than does the same intake from media with high bioavailability. The bioavailability of lead from normal infant diet is known to be very high (Alexander et al., 1974a,b; Ziegler et al., 1978; Ryu et al., 1983), with at least 40 to 50% of the dietary lead intake passing into the child's blood. See Section 4.1 for a discussion of bioavailability.

The main functions of the bioavailability menu are shown in Figure 2-7. The model calculates lead absorption from the gut as a function of two components. The *passive* component does not depend on lead concentration in the gut and is not saturable. The *facilitated or active* component may become saturated when the total concentration of lead in the gut from total gut intake by all media is sufficiently large, which is a kinetically *non-linear* absorption mechanism. The data entry Screen 2-14 allows the user to specify the parameters for intake from soil, dust, drinking water, diet, and alternate sources. The total absorption percentage is the sum of the passive and facilitated absorption components. The default value of absorption for alternate sources is 0%, which requires that the user must enter the bioavailibilty of any specific alternative source, such as lead-based paint.

The total absorption from any medium is then divided into two components, and the user specifies a small fraction of the total absorption percent for the passive or non-saturable (i.e., high-dose) component. The default is 20% of the total available for absorption. The percentage absorption in the larger saturable component is the remainder of the total available for absorption. For example, with a dietary lead intake of 50%, the absorption fraction for the passive component is 20% of 50%, or 10% of dietary lead intake, and the saturable component is 80% of 50%, or 40% of dietary lead intake.

2.2.2.7 Maternal-Fetal Lead Exposure (6)

The maternal lead exposure input parameter menu is shown in Screen 2-15. The lead is transferred from the mother to the fetus *in utero*. The lead that is stored in the tissues of the newborn child is calculated by entering the maternal blood lead value at birth (default = 2.5 μg/dL). The IEUBK model assumes that the infant's blood lead at birth is a fraction of the maternal blood lead level, and the amounts of lead in the blood and other tissues in the newborn infant are calculated so as to be consistent with concentration ratios observed in autopsies of newborn infants (Barry, 1981).

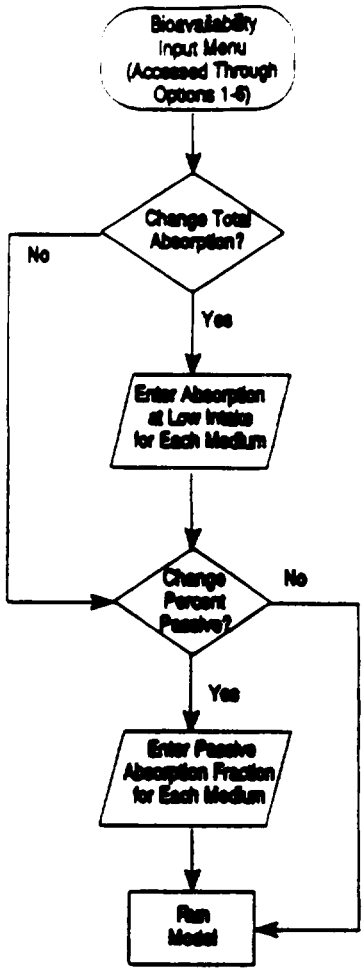
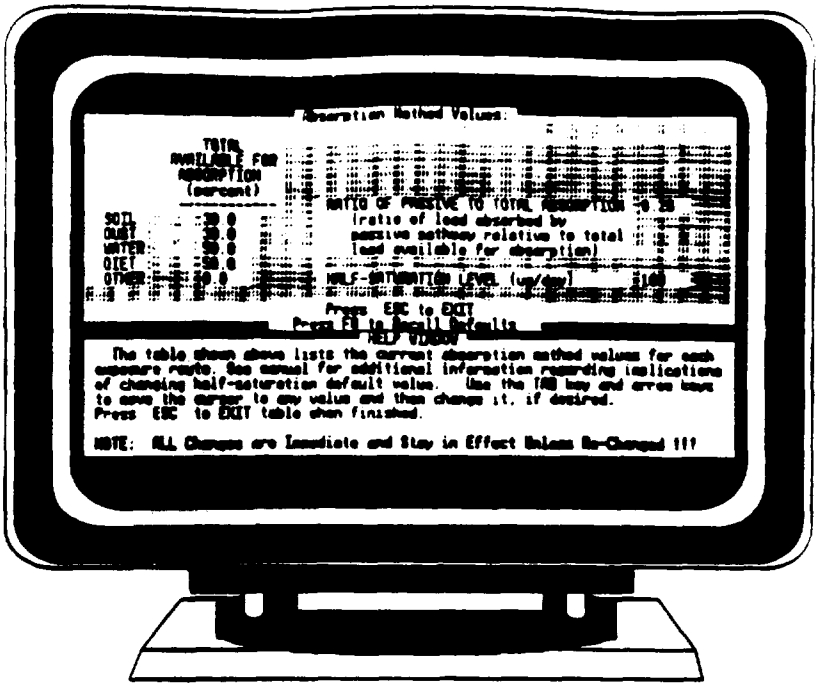


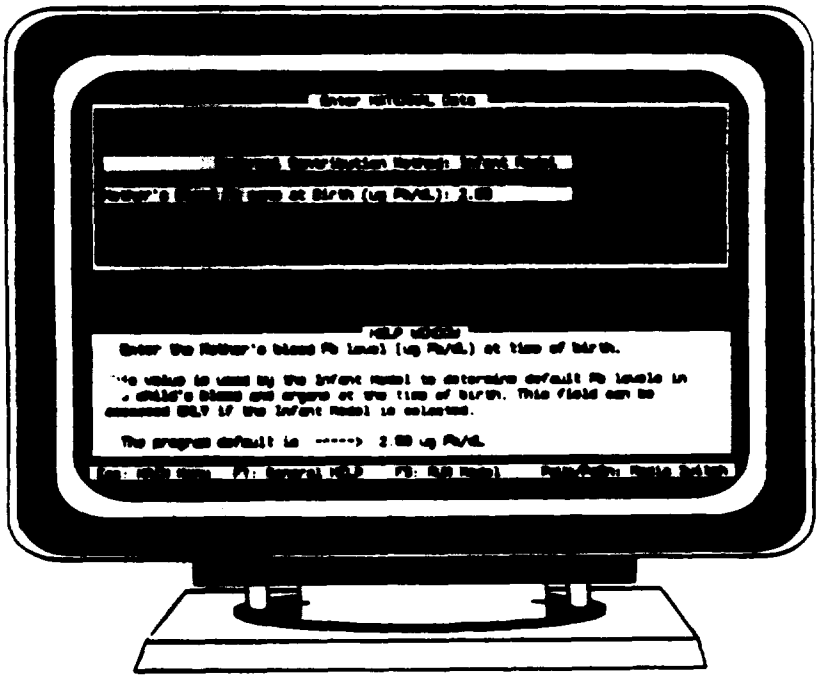
Figure 2-7. Decision diagram for the absorption/bioavailability menu options.

2.2.2.8 Save and Load Options

If the user wishes to use a certain set of model parameters as the starting point for another analysis, the parameter set created from use of Options 1 through 6 should be *saved* using the "S" option on the output menu accessed from the main menu, or the F6 option on any of the parameter input menus. The user may create an 8-character or shorter name for the file, which will be stored in the form [NAME].SV3. If a saved parameter set is needed later, it may be loaded from the "L" option on the Parameter Input Menu.



Screen 2-14. The absorption/bioavailability menu.

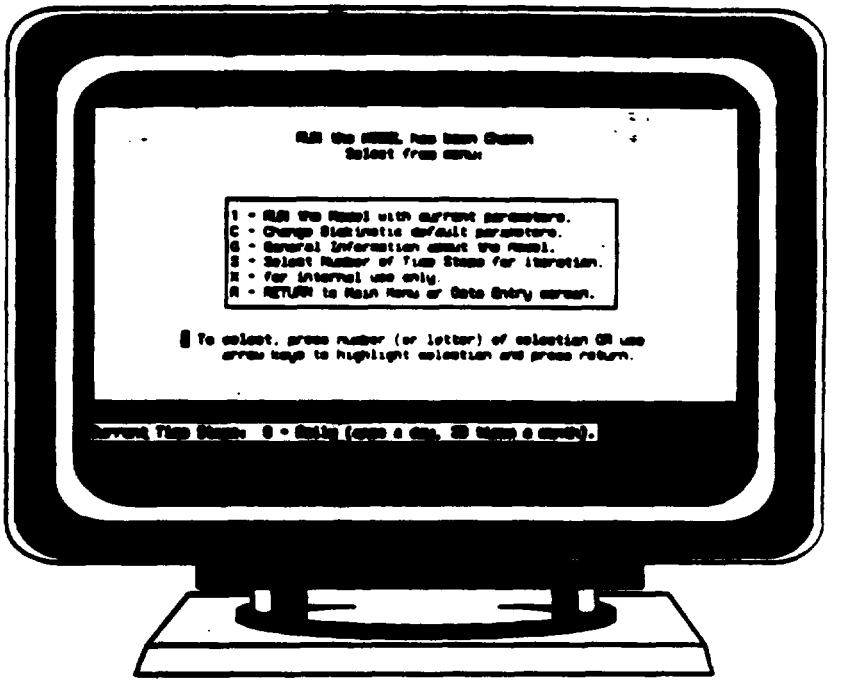


Screen 2-15. The maternal/fetal lead exposure menu.

2.2.3 Computation Menu

2.2.3.1 Run a Single Simulation of the Model (1)

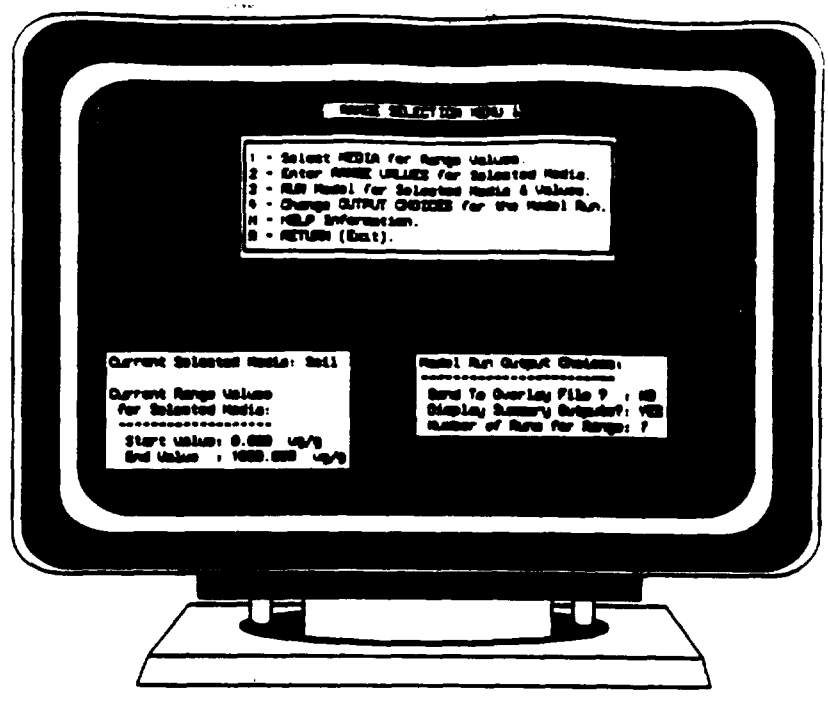
The menus for the Run command are shown in Screen 2-16. This option uses only the currently loaded parameter set. The user may view or change the time step for the numerical iteration. The default is four hours. We recommend setting the iteration time to the lowest convenient selection, and verifying all "important" solutions by rerunning the model with the shortest possible time step (currently 15 min). An output option (Option 2) allows plotting of results and calculation of probability of elevated blood lead.



Screen 2-16. Single simulation using the program processing menu.

2.2.3.2 Run Multiple Simulations of the Model for a Range of Media Lead (2)

The menus for the Multiple Run command are shown in Screen 2-17 and schematically in Figure 2-8. More detailed menus for range selection and output are shown in Screens 2-18 and 2-19. This option uses only the currently loaded parameter set, except that it repeats the run for each new value of a medium concentration (e.g., soil lead concentration) or intake (dietary lead as μg Pb per day). The user may view or change the



Screen 2-17. Multiple simulation using the program processing menu.

time step for the numerical iteration during the run step. We recommend verifying all of the "important" solutions by rerunning the model with the shortest possible time step (currently 15 min). Since only one medium can be changed in each use of the "2" option, the user who wants to look at a range of soil lead values should use the Multiple Source Dust option "3" and a user-specified dust lead to soil lead concentration ratio. Output data for plotting, with overlays of results at each concentration in the range, may be saved when the user creates RANGE#.LAY files.

2.2.3.3 Multiple Simulation Runs of a Medium To Find Concentration of Lead in the Medium That Produces a Specified Blood Lead (3)

This option is similar to Option 2. The menus for the Multiple Run command are shown in Screen 2-20 and schematically in Figure 2-8. This option uses only the currently loaded parameter set, except that it repeats the run for each new value of a medium concentration (e.g., soil lead concentration) or intake (dietary lead as μg Pb per day) until the specified age-dependent geometric mean blood lead level is achieved *exactly* by that concentration.

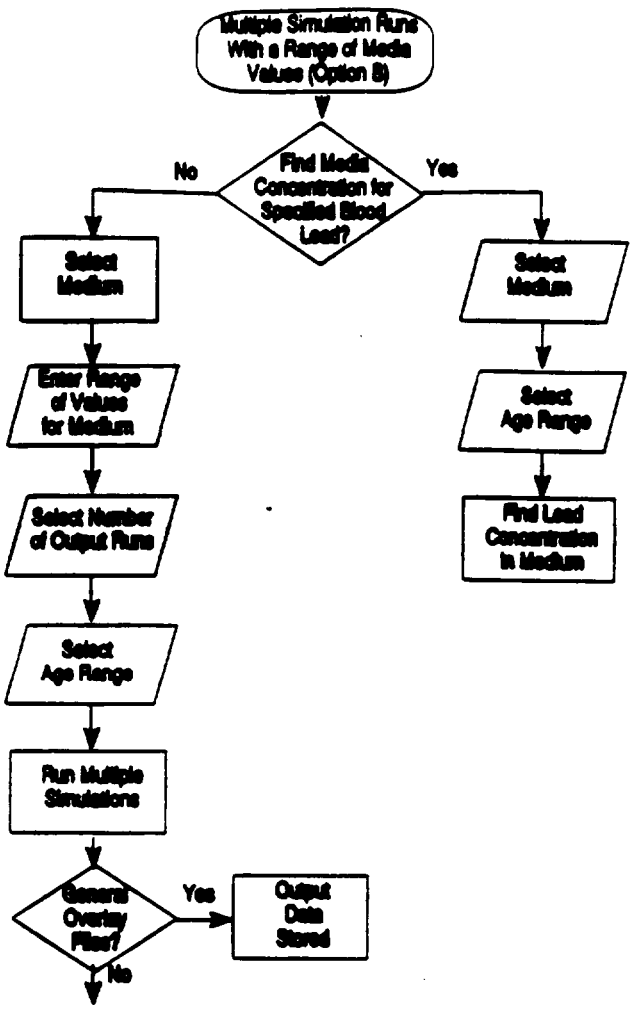
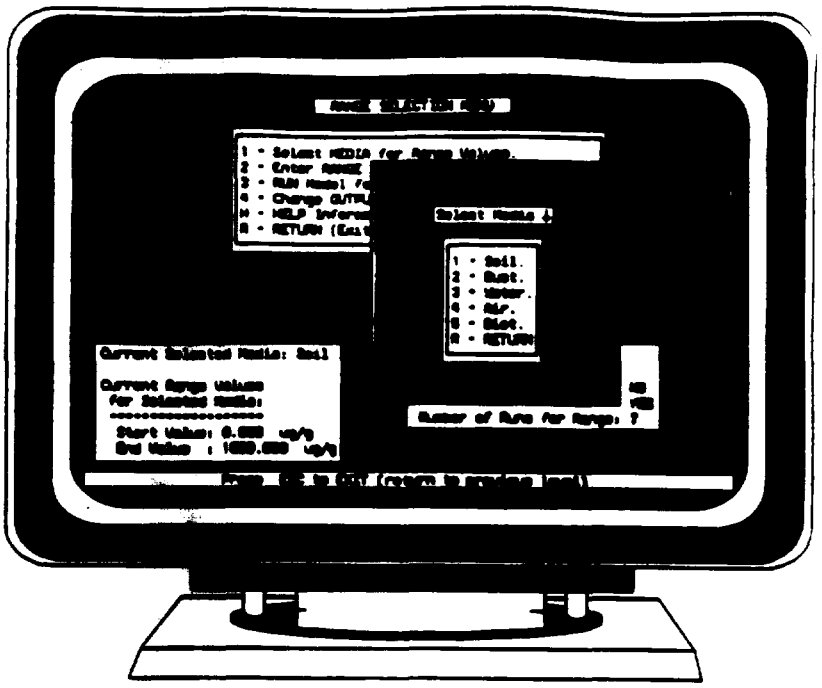


Figure 2-8. Decision diagram for the multiple simulation menu options.

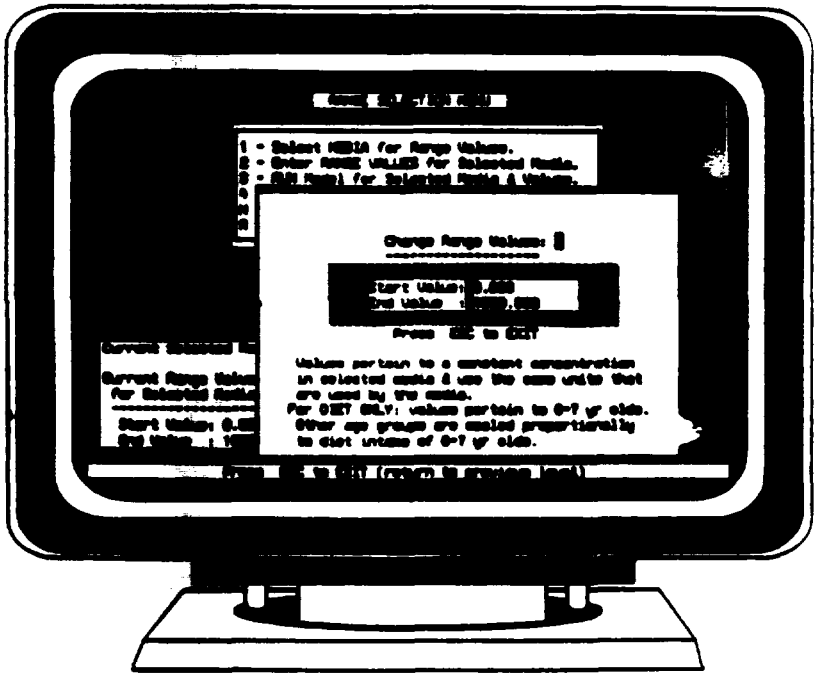
Since only one medium can be changed in each use of the Multiple Simulation Run "3" option, the user who wants to look at a range of soil lead values should use the Multiple Source Dust option "3" and a user-specified dust lead to soil lead concentration ratio. Output data for plotting may be saved when the user creates *.PBM files.

2.2.3.4 Batch Mode Multiple Simulation Runs Using Input Data Files (4)

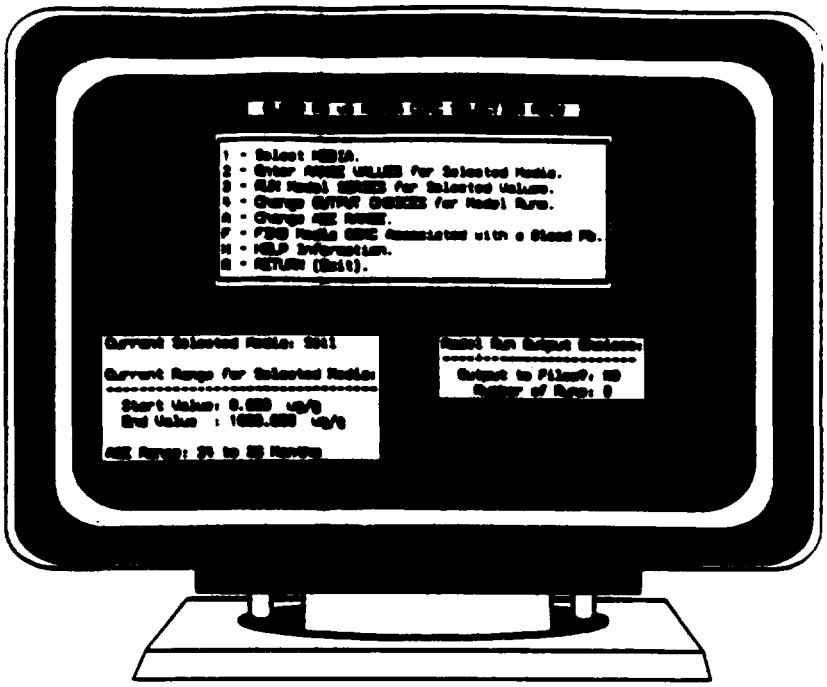
This option is similar to Option 2. The menus for the Batch Mode Run command are shown in Screen 2-21 and schematically in Figure 2-9. This option uses the currently loaded default parameter set, but repeats the run for using the new values for the five exposure



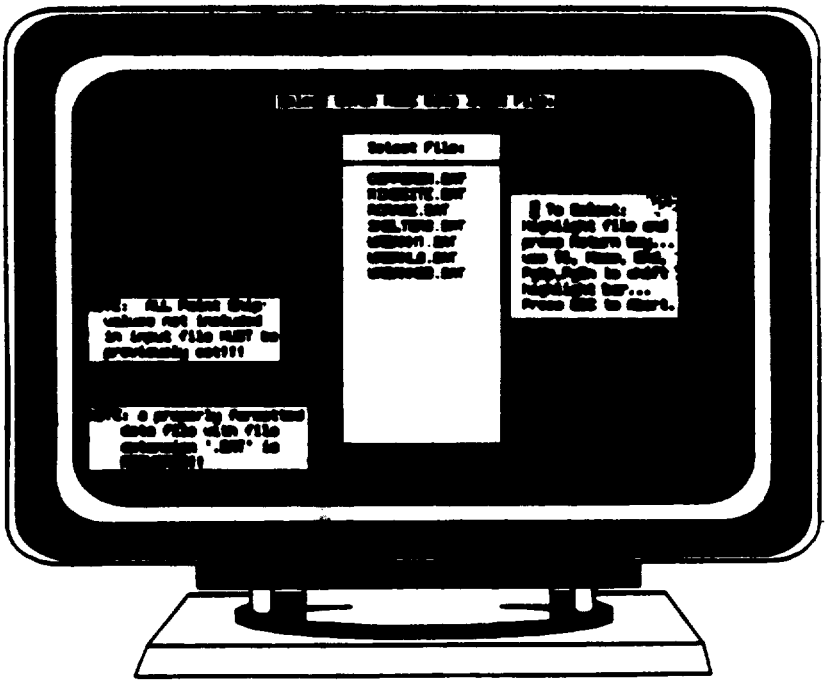
Screen 2-18. Selection of media for multiple range run.



Screen 2-19. Range selection during multiple processing.



Screen 2-20. **Using multiple simulation to find acceptable media concentrations for a predetermined blood lead concentration.**



Screen 2-21. **Running the model in batch mode.**

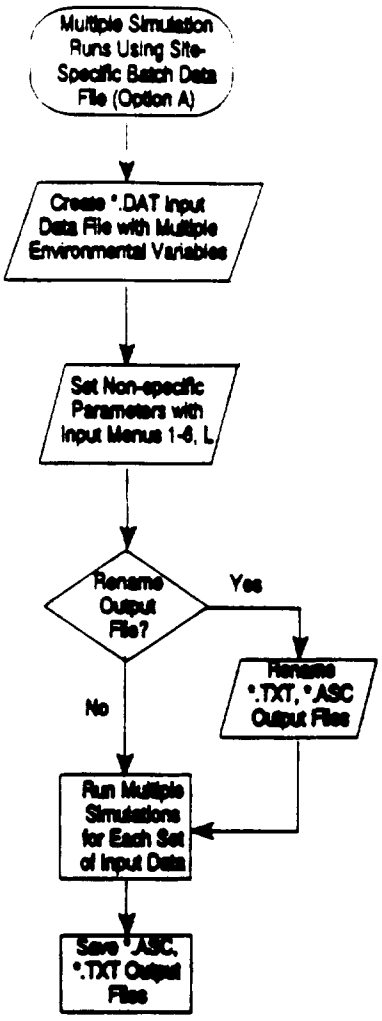


Figure 2-9. Decision diagram for the batch mode menu options.

parameters (soil concentration, dust concentration, drinking water concentration, air concentration and alternate source consumption) for each child in the data set. The input data are entered one line at a time from a data set with a specified list of input variables. These must be created by the user in a special *.DAT file in the Lead Model directory.

Each line of data *may* include:

The child code or case;

The "family" identifier for individuals at the same living unit;

An area, block, or neighborhood identifier;

Each line of data *must* include:

The child's age, in months, as of the end of the data collection period;

The soil lead concentration, in $\mu g/g$;

The house dust lead concentration, in $\mu g/g$;

The drinking water lead concentration, in $\mu g/L$;

The air lead concentration, in $\mu g/m^3$;

The alternate source intake rate, in $\mu g/day$;

The child's blood lead level at specified age, in $\mu g/dL$.

The child's age must be entered. Either a soil lead or a dust lead value is needed for the simulation, along with a stand-in value (imputation rule) if one of them is missing (for example, if the user does not fill in missing dust lead values, the current default is to replace a missing dust lead concentration by the soil lead concentration). The user may prefer to create an input data file with missing dust lead concentrations replaced by some fraction of the soil lead concentration. Missing values of air, water, and alternate lead are replaced by default values. If there is no actual child blood lead data, then Option 1 produces output data sets with *.ASC and *.TXT extensions that contain all of the input data, including imputed values, and predicted blood lead levels for each line of data.

The batch mode option can be used to perform statistical analyses of simulated community blood lead distributions, even without observed blood lead levels (for example, if an investigator has carried out a multimedia environmental lead study at a site, without blood lead data being collected). However, this option will be even more useful if blood lead data from a well-conducted study are available for model comparisons using statistical tests in Option 5. Output data files may be reviewed using Option 2, as demonstrated in later sections.

2.2.3.5 Statistical Analyses of Batch Mode Data Sets (4)

A set of statistical procedures for analyzing batch mode data sets exists as a separate module in the IEUBK Lead Model. Although the Option 1 data sets can be edited and used

in any other statistical programs the user may have available, we have included in Option 5 some of the most commonly used statistics, statistical hypothesis tests, and graphical data displays for comparing observed and modelled blood lead levels. We recommend using a variety of graphical and statistical techniques in evaluating the output of Batch Mode model runs. This will also be demonstrated in Section 5.

2.3 BUILDING AN EXPOSURE SCENARIO

2.3.1 Air Lead Menu

2.3.1.1 Default Air Lead Exposure Parameters

The default air lead concentration is 0.1 $\mu g/m^3$, which is approximately the average 1990 urban air lead concentration (U.S. Environmental Protection Agency, 1991b). During the period 1970-90, ambient air lead concentrations dropped drastically in the United States due to the phasedown of lead in gasoline (Figure 2-10). When adequate monitoring data exist to define concentrations higher or lower than the default outdoor lead concentrations, these should be used. Current air lead levels are low in most places in the United States, and do not require year-to-year specification. Elevated air lead levels have been reported around some point sources in the United States and Europe (Davis and Jamall, 1991) and lead modeling for changes in these sources requires year-by-year input data.

A constant air lead value larger than 0.1 $\mu g/m^3$ may be appropriate for assessment at locations in the vicinity of active point sources of lead emissions such as lead smelters or battery plants. In such cases, an appropriate estimate of annual average air lead concentration must be available.

An example of a striking increase over time was the air lead levels in Kellogg/Silver Valley, Idaho, following a September 1973 baghouse fire. These levels remained elevated for a sufficiently long time such that the use of these values in predicting blood lead concentrations for 1974-1975 from the Lead Model was justified (Agency for Toxic Substances and Disease Registry, 1988).

2.3.1.2 Ventilation Rate

The intake of air increases from infancy to adulthood. The range of values for child ventilation rates was established by EPA (U.S. Environmental Protection Agency, 1989a) as

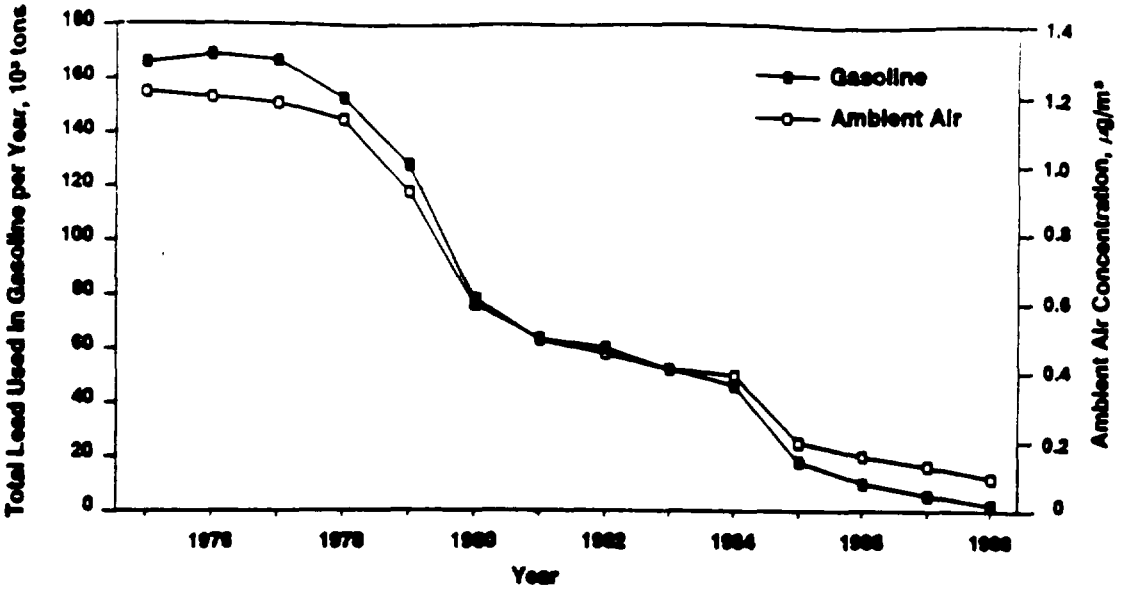


Figure 2-10. Historical relationship between lead in gasoline and lead in air in the United States.

Source: U.S. Environmental Protection Agency (1986), with updating.

2 to 3 m^3/day at age 0 to 1 years, 3 to 5 m^3/day at age 1, 4 to 5 m^3/day at ages 2 and 3, 5 to 7 m^3/day at ages 4 and 5, and 6 to 8 m^3/day at age 6. The Lead Model uses midrange values of 2, 3, 5, and 7 m^3/day at ages 0+, 1, 2 to 4, and 5 to 7 respectively. Children who exercise more than average will have a correspondingly greater intake, and those who are very inactive will have a lower ventilation rate. The higher intakes may be useful in modeling children who spend time at playgrounds or outdoor play areas near an air lead source. Changes in activity pattern can change ventilation rate in a child or in a neighborhood.

2.3.1.3 Indoor/Outdoor Activity Patterns

The range of values for outdoor time was established by EPA (U.S. Environmental Protection Agency, 1989a) as 1 to 2 h/day in the first year of life, 1 to 3 h at age 1, 2 to 4 h at age 2, and 2 to 5 h/day from ages 3 to 7. The default values in the Lead Model are 1, 2, 3, and 4 h/day at ages 0+, 1, 2, and 3+, respectively, roughly at the middle of these ranges. The outdoor air lead concentration provides a large part of the total air lead exposure, because the indoor air lead concentration is typically only about 30% of the outdoor concentration (U.S. Environmental Protection Agency, 1986). Site-to-site

differences may exist due to natural ventilation, climate, season, family activity, and community access to outdoor play activities.

2.3.1.4 Lung Absorption

The range of values for child lung absorption was established by EPA (U.S. Environmntal Protection Agency, 1989a) as 25 to 45% for young children living in non-point source areas, and 42% for those living near point sources. The default value used in the Lead Model is 32%. Changes in the source of airborne particulates may also affect lung absorption. No quantitative recommendations can be made.

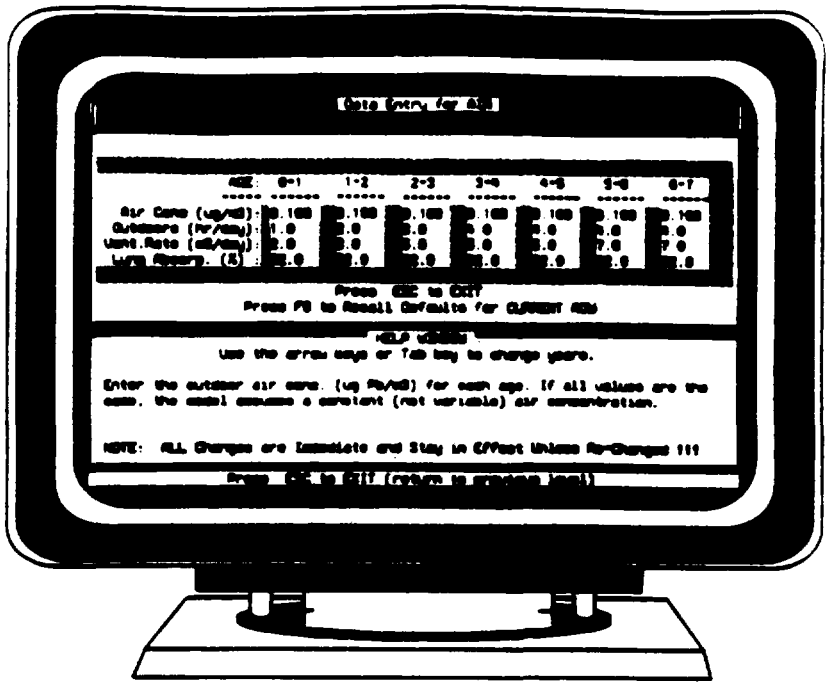
EXAMPLE 2-1: Characterizing Effects of Air Lead Phasedown on Inhalation Intake

If the Lead Model were to be used to estimate blood levels of children living in an urban area in previous decades, when the predominant sources of lead exposure for many U.S. children were air lead from combustion of leaded gasoline and dietary lead from lead-soldered food cans, it would necessary to use community air lead levels during that period of time. Representative values of air lead concentrations were presented in the EPA Air Quality Criteria Document for Lead (U.S. Environmental Protection Agency, 1986, Chapter 7, Table 7-2) for urban center or suburban locations in nine metropolitan areas for 1970 through 1984. The reductions in air lead from 1977 through 1988 attributable to the phasedown of leaded gasoline are quite evident in both urban centers and suburban areas. For example, for a retrospective estimate of blood lead levels in children in 1981, one would need to start with 1975 air lead levels to include prenatal exposure of children up to age 7 in 1981. Figure 2-10 shows that air lead exposure in 1981 would be at 0.48 $\mu g/m^3$, and so on. For a 5-year old child in 1981, air lead exposure, at age 0+ in 1975 is 1.2 $\mu g/m^3$, and so on. This adjustment in air lead concentration does not estimate the indirect effects of air lead changes on blood lead through gradual changes in soil and dust lead. This example is generic, not site-specific, however. The air lead data entry screen for children born in 1975 is shown in Screen 2-22.

2.3.2 Dietary Lead Menu

2.3.2.1 Total Dietary Lead Exposure

Data assembled from a variety of sources, including Market Basket Surveys (Pennington, 1983) and representing changes in consumer behavior over time, were used to construct dietary lead intake estimates as described in Chapter 7 of the EPA Air Quality Criteria Document for Lead (U.S. Environmental Protection Agency, 1986). The method is



Screen 2-22. Data entry for air. The user may input data from historical records of air lead concentrations on this screen.

based on U.S. FDA Market Basket samples in 231 food categories and has been updated to 1988 (U.S. Environmental Protection Agency, 1989a). Because two major sources of lead in food (lead-soldered cans and air deposition on food crops) have been greatly reduced or eliminated, dietary lead is believed to be relatively constant since 1990, especially for children under seven years.

Table 2-1 shows how estimated mean dietary lead intake depends on the child's age, and that this intake has changed very drastically with the near-elimination of lead solder from food cans and other food packaging in the United States since the 1970s. Where site-specific dietary levels are not available, it is recommended that the values from Table 2-1 be used for the appropriate years and ages, and that the most recent values (1988) be assumed for all future years. Seasonal effects are omitted here since the Lead Model uses annual values for dietary exposure parameters. For alternate exposure scenarios with seasonal intakes, the user may need to calculate time-weighted annual averages from seasonal data.

If the Lead Model is used in connection with historical exposures, for such purposes as model validation or retrospective dose reconstruction, the dietary intake data should be

TABLE 2-1. DIETARY LEAD INTAKE (μg/day) FOR U.S. CHILDREN BY AGE, FOR EACH YEAR FROM 1978 TO PRESENT

	Age						
	6-11 Mo	1 Year	2 Years	3 Years	4 Years	5 Years	6 Years
1978[1]	NE	45.8	52.9	52.7	52.7	55.6	NE
1979[1]	NE	41.2	48.0	47.8	47.8	50.3	NE
1980[1]	NE	31.4	36.9	36.9	36.9	38.7	NE
1981[1]	NE	28.3	33.8	33.7	36.8	35.8	NE
1982[2]	19.2	25.0	27.5	27.4	27.2	28.6	31.6
1983[2]	14.4	18.3	21.9	21.4	21.1	22.3	24.8
1984[2]	19.0	22.7	26.4	26.0	25.7	27.1	29.9
1985[2]	10.2	10.6	12.3	11.9	11.8	12.4	13.6
1986*	7.9	8.2	9.4	9.1	8.9	9.4	10.3
1987-Present[3]	5.5	5.8	6.5	6.2	6.0	6.3	7.0

NOTES: NE = Not estimated.
1 = Estimated by J. Cohen and D. Sledge, Table A-2 (U.S. Environmental Protection Agency, 1989a).
2 = U.S. Environmental Protection Agency (1986), updated with data from the FDA Market Basket Survey.
3 = Average of 1986 Q4 through 1988 Q3. Further decreases in food lead concentrations since 1987 are believed to be negligible.
* = Linear extrapolation between 1985 and 1987.

adjusted to the year when the data were collected. For prediction in future years, the most recent default value for each age may be used.

2.3.2.2 Dietary Lead Exposure by Additional Pathways

For some children, there are important dietary lead sources that are not characterized by the FDA Market Basket Survey data summarized in Table 2-1. Child-specific or site specific data will be needed to verify these alternative dietary lead sources. Local sources of fruit and vegetables are used in many small towns and in rural areas. Some individuals obtain much of their produce from their own gardens. If the local or home-grown produce is grown in soils with high concentrations of lead, or if the edible leafy portions are contaminated by airborne lead particles, then some fraction of the environmental lead may be added to the child's diet. The additional intake of lead in diet may become important if the

environmental lead concentrations are sufficiently high. This was important in evaluating the Bunker Hill Superfund site in Kellogg ID and was included in the Risk Assessment Data Evaluation Report (the "RADER") prepared by EPA (U.S. Environmental Protection Agency, 1990c). Additional pathways of dietary lead exposure are discussed in Example 2-2.

Dietary lead exposure is the product of the amount of food consumed in each category and the concentration of lead in the food item. Normal intakes are reported by Pennington (1983). To adjust for home gardens, a fraction of this intake may be allocated by the Alternate Diet Entry Menu to local produce, and the rest to Market Basket produce that is not grown locally.

Local game animals feeding on plants contaminated by lead in soil may also have elevated lead concentrations. Lead contamination of rivers and lakes by deposition and by erosion of leaded soils may also increase lead concentrations in local fish. Some rural families may use hunting and fishing as a significant supplement or even as their primary source of animal protein. See Baes et al. (1984) for a comprehensive approach to estimating pathways of trace elements in the food chain. A fraction of the meat and fish intake may be allocated by the Alternate Diet Entry Menu to local game and fish.

Other consumer products may have nontrivial potential for dietary lead exposure. These include lead-glazed or soldered cooking and food preparation utensils, ethnic or regional preferences for food products with high lead content, and the use of oral ethnic medicines such as "empacho" or "azarcon" that have high concentrations of lead and are known to have caused cases of acute lead poisoning in children (Trotter, 1990; Sawyer et al., 1985). No general recommendations about parameter values for these sources of lead can be made at this time. Approximate intake for oral medicines may be estimated from recommended or customary doses for young children.

EXAMPLE 2-2: Characterizing Indirect Dietary Lead Intakes for an Old Lead Smelter Community

Some data from the Human Health Risk Assessment (Jacobs Engineering, 1989) and the RADER for Kellogg ID may be useful (U.S. Environmental Protection Agency, 1990c). Table 2-2 shows that a large percentage of the population uses local produce, that the use of local produce increases toward the more rural Pinehurst area but the lead concentration decreases, and that the lead levels in local produce in 1983 were enormously higher than in

TABLE 2-2. ESTIMATES OF LEAD INTAKE FROM CONSUMPTION OF LOCAL PRODUCE BY CHILDREN, AGES 2 TO 6 YEARS, IN KELLOGG, IDAHO

Area	Percent Using Local Produce	Number of Gardens	Consumption (g/day) Leafy*	Root*	Concentration (μg/g wet wt.) Leafy	Root	Intake (μg/day) In Summer**
Smelterville	16%	2	25	15	6.1	4.5	220
Kellogg	36%	17	25	15	6.1+	4.5+	220+
Pinehurst	46%	20	25	15	3.5	2.2	121
National Market Basket	–	–	25	15	0.017	0.041	1

NOTES: *Leafy vegetables are lettuce and spinach. Root vegetables are carrots and beets.
+Average of Kellogg and Smelterville.
**Annual average is 1/4 of this.

Source: RADER Tables 5-8 and 5-4 (U.S. Environmental Protection Agency, 1990).
Jacobs Engineering (1988) Table 7-16.

the National Market Basket samples for the same period (1982 to 1984). The calculated increment of daily dietary lead intake during the summer months was 220 μg/day in the report. However, for the purposes of this example, we will assume that this total consumption occurs over the course of the year and includes fresh as well as frozen or canned produce to give an annual average increment of 55 μg/day.

Table 2-3 shows that the lead concentration in fish in nearby Lake Coeur d'Alene in 1985 was much higher than in the Columbia River and higher than fish at the average National Pesticide Monitoring Station lead concentration for 1976/1977. A moderate rate of consumption is two 2-oz fish portions per week, or 114 g/week = 16 g/day on average. The incremental intake from local fish is equal to the concentration difference, 0.80 to 0.34 = 0.46 μg/g times 16 g/day = 7.5 μg/day.

Screen 2-23 shows dietary lead intakes for a typical child born in 1983, and Screen 2-24 shows the extra exposure for intake from contaminated fish.

2.3.3 Drinking Water Lead Exposure Menu

2.3.3.1 Drinking Water Lead Default Exposure Parameters

Water sampling methods may be as first draw standing samples, partially flushed samples, or fully flushed samples. The highest lead concentrations at the tap are usually obtained for lead pipes, lead-alloy solder on copper pipes, or lead-alloy brass faucets and

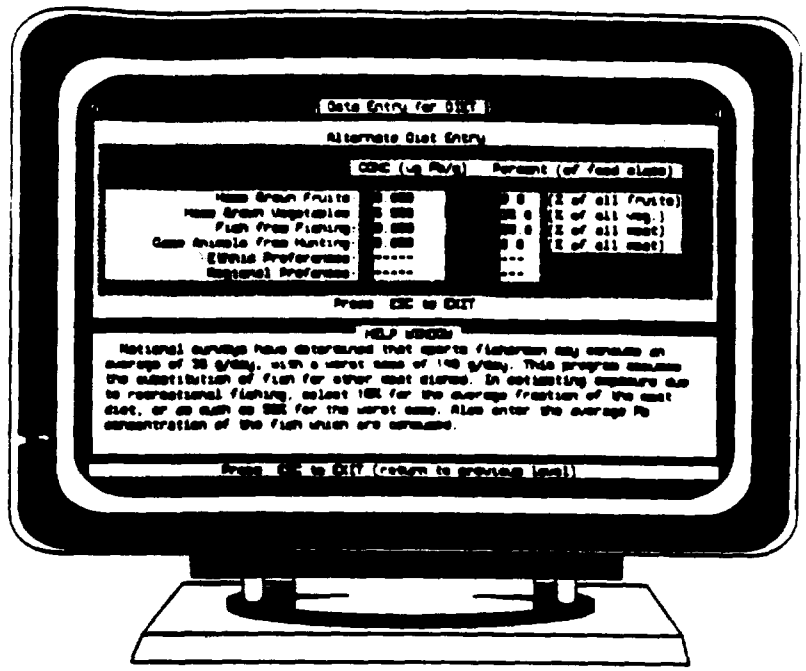
TABLE 2-3. ESTIMATES OF LEAD INTAKE FROM CONSUMPTION OF LOCAL FISH BY CHILDREN, AGES 2 TO 6 YEARS, IN KELLOGG, IDAHO

Source	Concentration (μg/g wet wt.)	Fish Consumption* (g/day)	Lead Intake** (μg/day)
Lake Coeur d'Alene (1985)	0.80	16	13.0
Columbia River (1986)	0.34	16	5.5
National Pesticide Monitoring Stations (1976-1977 August)	0.34	16	5.5

NOTES: *Two-ounce portions, twice a week.
**For annual average, multiply by fraction of year when local fish are consumed.

Source: RADER Tables 5-8 and 5-4 (U.S. Environmental Protection Agency, 1990).
Jacobs Engineering (1989) Table 7-16.

Screen 23. Using dietary lead intake for a child born in 1983 (see Example 2-2).



Screen 24. Using dietary intake from local vegetables and fish in Kellogg (see Example 2-2).

fittings in contact with corrosive water for several hours. The new EPA National Primary Drinking Water Regulation for Lead (NPDWR) requires public water systems to collect first draw samples, standing a minimum of 6 h, from a sample of homes targeted as potentially at risk. Water lead concentrations can be significantly different for different sampling protocols, depending on the sources of lead in water drawn through the tap. First draw samples generally have higher lead concentrations than flushed samples. The typical effects of different water sampling procedures are discussed in the Sampling Manual that is to accompany this model.

Drinking water lead concentrations in the Lead Mode are held constant during the entire seven years of the child's exposure. In the Case Studies below, household-specific water lead concentrations are used. If no household-specific or relevant community water lead data are available, we recommend using the default value of 4 μg/L.

If a substantial fraction of the child's activity is spent outside the home, it may be useful to separate drinking water exposure into primary residence and secondary residence or daycare. A large number of U.S. children spend time during the weekday at daycare centers

or secondary residences. If adults and older children in the household are either at work or at school during the day, there may be two stagnation periods for drinking water during the day—overnight, and midday. In this case, a larger fraction of the child's water lead exposure can occur at the higher "first-draw" concentrations. Some exposure scenarios are discussed by Marcus (1991) in evaluating the risk from lead leached out of newly installed brass faucets. The default scenario is defined by setting 50% of the child's water intake to household first-draw consumption. The remaining intake consists of partially flushed intake inside the home (35%) and water consumed outside the home (15%). The total intake of lead in drinking water would then be:

$$\text{PbW} = 0.5 \times \text{PbW (first draw)} + 0.35 \times \text{PbW (flushed)} + 0.15 \times \text{PbW (fountain)}.$$

There is no general rule for estimating the amount of water ingested from water coolers in day care centers or other non-home locations. Since the child's activities outside the home are likely to be different than inside the home, it is unlikely that the ratio of non-home to home water intake is proportional to the amount of time spent away from the home versus at home. Two drinks per day, each about 60 mL (2 oz) or 120 mL, is a reasonable upper limit for day care intake. The default is 15% of the daily tap water intake, which ranges from 75 mL at age 1 year to 90 mL at age 6 years.

2.3.3.2 Alternate Drinking Water Exposure by Age

The default values in the IEUBK model (Table 2-4) are taken from the U.S. EPA Exposure Factors Handbook (U.S. Environmental Protection Agency, 1989c). A survey of drinking water consumption in U.S. children was reported by Ershow and Cantor (1989) in a study for the National Cancer Institute. These values have been smoothed and disaggregated into yearly values shown in Table 2-4. The range of values from the Ershow-Cantor data in Table 2-5 show that the default values for the IEUBK model are similar to but somewhat lower than the median values, but also contain information about the percentiles of the distribution of tap water intake, about gender differences in intake and other factors that you may find useful. A plausible scenario for elevated exposure to lead in drinking water would be to use larger tap water intakes, such as the 90th percentile values in Table 2-5. Note that for children receiving formula reconstituted with tap water, consumption of tap water would be much higher, perhaps closer to one liter per day. In an assessment addressing risks from lead in drinking water, the exposure to infants consuming reconstituted formula requires specific attention.

TABLE 2-4. AVERAGE DAILY WATER INTAKE IN U.S. CHILDREN

	Ershow-Cantor Study*				IEUBK Model**	
	Total (L/day)		Tap (L/day)			
Age (Months)	M	F	M	F	Age (Mo)	Tap Water Intake (L/day)
0-5	0.992	1.035	0.250	0.293	0-5	0.20
6-11	1.277	1.238	0.322	0.333	6-11	0.20
					12-23	0.50
					24-35	0.52
12-47	1.409	1.300	0.683	0.606	36-47	0.53
					48-59	0.55
					60-71	0.58
48-84	1.551	1.488	0.773	0.709	72-84	0.59

*Ershow and Cantor (1989).
**U.S. Environmental Protection Agency Exposure Factors Handbook (1989c).

TABLE 2-5. TAP WATER INTAKE (L/day) BY AGE CATEGORY

Age Category		Percentiles		
(Months)	Mean	10	50 (Median)	90
0 - 5	0.27	0	0.24	0.64
6 - 11	0.33	0	0.27	0.69
12 - 47	0.65	0.24	0.57	1.16
48 - 84	0.74	0.30	0.66	1.30

Source: Table 2-5, Ershow and Cantor (1989).

2.3.4 Soil/Dust Lead Exposure Menu

One of the most important uses of the IEUBK model is to compare risks among alternative soil lead and dust lead exposure scenarios. Many of these scenarios arise in assessing exposure reduction strategies. For example, in evaluating soil lead abatement at a particular residential yard, we might be interested in the following sequence of comparisons:

(1) Calculate the risk of an elevated blood lead level for the present soil and dust lead levels;

(2) Calculate the risk of an elevated blood lead level for the replacement of soil lead with soil having a lower lead concentration, along with cleaning up household dust;

The first scenario describes risk to occupants with present exposure levels. The second scenario describes risk to occupants in the distant future after lower new lead levels have been achieved by abatement. The IEUBK model can accept input data describing both of these exposure scenarios.

2.3.4.1 Soil and Dust Lead Default Exposure Parameters

The natural concentration of lead in soil, from weathering of crustal materials, is estimated as about 10 to 25 μg/g. A plausible urban background is 75 to 200 μg/g (U.S. Environmental Protection Agency, 1989a; HUD, 1990).

It is expected that lead concentrations in undisturbed soils may persist for many thousands of years. However, urban areas are hardly undisturbed environments and available data (von Lindern, 1991; Jacobs Engineering, 1990) suggest that near-surface soil lead concentrations may decrease by a few percent over a decade or so. It is usually adequate to assume a constant soil lead concentration unless soil abatement is included in the exposure scenario.

It is also possible that the soil becomes recontaminated over time, for example if surface soil is abated and then is recontaminated by ongoing atmospheric lead deposition from non-abated sites near by or by contamination from deteriorating exterior lead-based paint. Changes in soil concentration can be incorporated on an annual basis in developing the exposure scenario. This is done with the Option "2" on the Soil/Dust Data Entry Menu.

2.3.4.2 Exposure to Soil and Dust

The default value for total intake of soil and dust depends on age, and ranges from 85 to 135 mg/day. These values are within the ranges identified in the OAQPS staff paper that supported the first UBK model and have been reviewed by the EPA Clean Air Science Advisory Committee. Recent investigations by Binder et al. (1986), Clausing et al. (1987), Calabrese et al. (1989, 1991b), van Wijnen et al. (1990), and Davis et al. (1990) apply the trace element approach to quantify ingestion rate. These investigations currently constitute the most appropriate basis for estimating the quantity of soil ingested. The results are summarized in Table 2-6. The van Wijnen et al. data are discussed in Section 2.3.4.4. It is likely that the intake rate depends on the child's age, activity pattern, and the total

TABLE 2-6. DAILY INTAKE OF SOIL AND DUST ESTIMATED FROM ELEMENTAL ABUNDANCES

Study	Element	Soil/Dust Intake, mg/day		
		Median	Mean	Maximum
Davis et al. (1991)	Al	25	39	904
Ages 2-7 years	Si	59	82	535
	Ti	81	246	6,182
Calabrese et al. (1989)	Al	30	154	4,929
Ages 1-4 years	Ti	30	170	3,597
	Y	11	65	5,269
	Zr	11	23	838
Binder et al. (1986)	Al	121	181	1,324
Ages 1-3 years	Si	136	184	799
	Ti	618	1,834	17,076
Clausing et al. (1987)	Al	92	232	979
Ages 2-4 years	Ti	269	1,431	11,620
	AIR	106	124	302

AIR = Acid Insoluble Residue.

accessible dust and soil in the environment. It is *recommended* that soil and dust intake be defined by an age-dependent scenario shown in Table 2-7, as reviewed by the Clean Air Science Advisory Committee (U.S. Environmental Protection Agency, 1990b).

Two of the studies, Davis et al. (1991) and Calabrese (1989), measured the dietary (including medication) intake of the trace elements and subtracted this quantity in estimating soil ingestion. These studies therefore provide the most complete quantitation of ingestion. Because Binder et al. (1986) did not measure dietary intake, the results for this study are likely to provide an upper bound on ingestion among those subjects. Van Wijnen et al. (1990) did not measure dietary intake but attempted to compensate for this approach by using the lowest observed tracer result for each child and subtracted out a value obtained for hospitalized children who were assumed not to ingest soil or dust. The combination of these two techniques may lead to a downward bias in ingestion estimates.

TABLE 2-7. AGE-SPECIFIC SOIL AND DUST INTAKE

Age (Years)	Intake (g/day)	Adopted for Guidance Manual
0 - <1	0 - 0.085	0.085
1 - <2	0.080 - 0.135	0.135
2 - <3	0.080 - 0.135	0.135
3 - <4	0.080 - 0.135	0.135
4 - <5	0.070 - 0.100	0.100
5 - <6	0.060 - 0.090	0.090
6 - <7	0.055 - 0.085	0.085

Source: U.S. EPA (1989a).

The reader should also note that there are statistical problems in interpreting an observed median value from these studies. For example, in a population of children who all ingested very small amounts of soil on most days but occasionally ingested larger quantities, the median from a short term measurement study will be below the average daily quantity ingested by any of the children. The mean value is not subject to this bias, and therefore is judged to be a more meaningful measure of ingestion.

It should be noted that the 200 mg/d ingestion value presented in Superfund guidance can be supported as, roughly, an upper bound on mean ingestion considering the values seen in different ingestion studies. The values recommended for use in the model (85 to 135 mg/d) represent a more central value within the range of values seen in different studies.

The smaller study of Clausing et al. (1987) used methods similar to the later study of van Wijnen et al. The values shown for soil ingestion in Table 2-7 are uncorrected for dietary intake. The paper presents additional estimates using acid insoluble residue and tracer excretion by hospitalized children.

2.3.4.3 Sources of Dust Exposure

Contribution from Atmospheric Deposition and Soil

We recommend collecting household dust data. If that has not been done, then Option 3 may be used to estimate dust lead concentrations. The OAQPS Exposure Analysis and Methodology Validation (U.S. Environmental Protection Agency, 1989a), used for the earlier version of the model on which the current IEUBK Model is based, calculates the

contribution of atmospheric deposition and soil to house dust by linear regression models between dust lead, soil lead, and air lead. There is a relationship between dust lead concentration in $\mu g/g$ (denoted as PbD), soil lead concentration in $\mu g/g$ (denoted as PbS), and air lead concentration in $\mu g/m^3$ (denoted as PbA). In a number of studies, statistically significant relationship of the form:

$$PbD = \beta_O + \beta_S\ PbS + \beta_A\ PbA$$

This equation suggests that house dust lead concentration consists of three components: a soil component, which is the fraction β_S of the soil concentration, an air component, consisting of a coefficient β_A relating $\mu g/g$ lead in dust to $\mu g/m^3$ of lead in air, and a third component of β_O coming from unidentified sources.

As a default value in the model, we used $\beta_A = 100\ \mu g/g$ per $\mu g/m^3$ based on several analyses. We recommend a default soil-to-dust coefficient of 0.70, which represents some real sites where soil is a major contribution to household dust. The reader should be aware that other values have been identified for other site-specific exposure scenarios.

Dust Lead Increment from School Dust

Dust ingestion while at school may be significant, depending on the amount of exposure on the floor or playground. While the IEUBK model deals primarily with preschool children, some children may be in school and subject to a more structured regimen of hygiene and reduced dust exposure. The amount of dust ingested and its implicit fraction of total dust ingestion is not necessarily proportional to length of time at the facility. Hygiene and dust loading are additional predictive factors. Playground geometric mean dust lead levels of 170 - 3,700 $\mu g/g$ were reported by Duggan et al. (1985) in a sample of 11 British schools.

Dust Lead Increment from Day Care

Dust ingestion while at daycare (including nursery school and kindergarten) may be significant, depending on the amount of exposure on the floor or outside play area. Dutch children who spent a considerable amount of time at a daycare center were known to ingest a large quantity of dust and soil, although apparently much less in rainy weather than in good weather (van Wijnen et al., 1990).

Dust Lead Increment from Second Home

Children often spend several hours per day in the home of a relative or in an informal daycare setting. Dust exposure information can often be collected and used in the same manner as for the primary home.

Dust Lead Increment Remaining from Primary Residence

When the Multiple Source Analysis option is selected on the Soil/Dust Data Entry Menu, the IEUBK model offers the opportunity to change the soil and air parameters of the regression equation set at 0.70 and 100, respectively as default values. The selection of a default value for the soil-to-dust coefficient was based on empirical data. In sites where soil to-dust coefficients have been measured and where paint does not contribute greatly to dust, the range was from 0.09 to 0.85. Among the sites where soil-to-dust coefficients have been measured are the following: East Helena, 0.85 (0.81 and 0.89); Midvale, 0.70 (0.68, 0.72); Butte, 0.26; and Kellogg, 0.09. Recent data suggest the coefficient decreases over time at some sites where major sources of soil lead deposition are no longer active. The user is cautioned, however, that the contribution of soil to dust concentration varies greatly from site to site, and site-specific soil and dust data should be collected for use in the model. The user may choose to enter values for alternate sources of dust, including both an estimate of concentration ($\mu g/g$) and relative contribution (%) for each source. Of the five alternate sources, two (secondary occupational dust and lead-based paint in home) represent contributions to house dust lead within the home, and three (dust at school, dust at daycare, and second home dust) represent exposure outside the primary home. If no selection is made from any of these five, the house dust concentration remains as calculated from the linear equation. If any of the five options are selected, this percentage is subtracted from the house dust component, the contribution from all sources is calculated, and the average is shown on the Multiple Source Average line of the Soil/Dust Data Entry Menu. This line appears only if the Multiple Source Option is selected.

2.3.4.4 Fraction of Exposure as Soil or Dust

We recommend using the default assumption that 45% of the total dust intake is derived from soil. The ratio of soil intake to dust intake is not simply proportional to the ratio of the number of waking hours that the child spends outdoors versus indoors. Children spend only 15 to 30% of their waking hours playing outside but are more likely to be in contact with bare soil areas, in locations with large amounts of accessible loose particles, and are likely to wash their hands less often than when they are indoors. The default 45/55 ratio in the model represents our best judgement of a properly weighted ratio for this parameter.

The issue of intake of soil and dust has not been properly resolved in the scientific literature. The distinction is important because there is some indication that even if soil lead is the principal source of dust lead, there may be chemical or physical differences between soil and dust that may affect bioavailability. Calabrese (1992) has found that most of the soil and dust intake in a soil pica child was in the soil component, but this is hardly representative of a larger population that may have large differences in relative exposure to soil and dust.

Section 2.3.4.4 discusses the option to select the amount of dust and soil consumed by the child each day. The default values are age weighted from 85 to 135 mg/day, and this dust is ingested either during kitchen preparation of food or by hand-to-mouth activity during indoor and outdoor play activity. This section discusses the option to allocate a portion of the ingested dust to dust derived from soil that is ingested during outdoor play activity. This distinction is important when there are differences between the bioavailability of dust derived from soil and dust in the home, and when there are large differences in the concentration of dust from the two sources. When house dust is thought to be mostly of soil origin and each are expected to have similar bioavailability, the designation of this fraction is a moot point. It is in cases where house dust differs significantly from soil derived dust that the soil/dust ratio becomes important. One example might be the presence of interior lead-based paint. In this case the parameter can be effective in separating soil derived dust and paint derived dust into two components where both the amount ingested and percent absorbed can be correctly input into the model.

There is some evidence that the soil intake is very responsive to exogenous factors, such as weather and location. Data reported by van Wijnen et al. (1990), summarized in Table 2-8, show the lowest soil and dust intakes at daycare centers occurred in rainy weather, when the children had the least amount of outdoor activity.

There is an implicit assumption that the exterior dust that a child ingests during outside play activity is derived completely from soil, and we use soil as a surrogate for exterior dust exposure. These intakes were measured during a 3 to 5 day sampling period, when soil and dust intake estimates ranged from 33 to 88 mg/day for children aged 1 to 2 years and from 12 to 62 mg/day for children older than 3 years. The intake of soil and dust is describe in detail in Section 2.3.4.2.

In the absence of any better data, we have reanalyzed and reinterpreted the van Wijnen et al. data based on the assumption that the rainy-weather intake is only interior dust, and

TABLE 2-8. MINIMUM PERCENTAGE SOIL INTAKE AS A FUNCTION OF AGE IN DUTCH CHILDREN IN DAYCARE CENTERS[a]

	Estimated Geometric Mean LTM, mg/day		
Age (years)	Good	Rainy	Difference (mg/d)
< 1	102 (4)	94 (3)	8
1 - < 2	229 (42)	103 (18)	126
2 - < 3	166 (65)	109 (33)	57
4 - < 5	132 (10)	124 (5)	8

[a]Minimum daily ingestion of acid insoluble residue or other tracers, denoted LTM (Limiting Tracer Method) from Table 4 in Van Wijnen et al. (1990). Number of children shown in parenthesis.

that the good-weather intake is both interior and exterior dust although probably with a smaller amount of interior dust than in rainy weather. The authors also made the distinction between soil and dust in their discussion of the study. For our reanalysis, we took the rainy-weather intake by age as dust and the good-weather intake as soil plus dust, to estimate an age-related difference of 8 to 126 mg/day soil (Table 2-8). The difference between LTM during good weather and LTM during mostly rainy weather is believed to be a lower bound on the soil intake. The combined intakes of soil and dust estimated by other authors are of a similar order of magnitude, such as the median soil and dust intake of 25 to 81 mg/day found by Davis et al. (1991) for children of ages 2 to 7 years in Richland-Pasco-Kennewick, Washington. We therefore assume that a substantial fraction of the combined soil and dust intake in U.S. children is in the form of soil, as suggested by the large difference in Table 2-8 between good and rainy weather intakes, and a substantial fraction is in dust, as suggested by the large intake during rainy weather, in Table 2-8. The minimum intake, denoted LTM for Limiting Tracer Method, has not been corrected for food intake. However, it is likely that the differences between LTM intakes do not depend on food intake.

2.3.4.5 Bioavailability of Lead in Soil and Dust

The current assumption in the Lead Model is that 30% of dust and soil lead intake is absorbed into the blood. This is assumed to be partitioned into a nonsaturable component of 6% and a saturable component of 24%. Some investigators (Steele et al., 1990) argue that the bioavailability of lead in soil from some old lead mining sites is much less than that of dissolved lead salts for several reasons: (1) large lead particles may not be completely dissolved in the GI tract; (2) the solubility of chemical species commonly found in mine wastes, particularly lead sulfide, is much lower than that of other lead salts. These

hypotheses are based on studies in small laboratory animals such as rats (Barltrop and Khoo. 1975; Barltrop and Meek, 1979), and while the results may be qualitatively relevant to humans, it is not clear how they should be extrapolated to humans or to other large animals with similar physiological properties such as baboons or swine.

2.3.5 Alternate Source Exposure Menu

One possible use of the Alternative Source Exposure Menu is the direct ingestion of chips of lead-based paint (LBP). The user might assume that a child with pica for paint ingests one paint chip per day. If this chip weighs 0.3 grams and contains lead at 10% (100,000 μg/g), then the calculated ingestion is 100,000 μg/g $\times$ 0.3 g/day, or 30,000 μg/day each day for a year. Note that this exposure would be in addition to exposure to lead-based paint in housedust, which is Option 4 in the Multiple Source Menu of Soil and Dust. The limited information available on the bioavailability of lead in paint chips suggests that at doses this high, absorption mechanisms may be largely saturated (Mallon, 1983), which would indicate appropriate adjustments in bioavailability. The user is referred to Section 4.7, and is encouraged to review the literature on this topic prior to making a risk assessment decision. Similar calculations can be made for the ingestion of soil or other non-food items.

2.4 STARTING AND RUNNING THE MODEL

2.4.1 Loading and Starting the Model

The IEUBK Model is a stand alone software package that requires only an IBM compatible PC with DOS. The diskette accompanying this manual contains the following files:

LEAD99d.EXE (the main program file)
PBHELP99.HLP (a help file)

PBSTAT.EXE (the statistical package)

Several *.BGI files (for graphic output)

One or more EXAMPLE*.DAT (sample data sets)

Copy all files into a directory of your choice, then type LEAD99d at the DOS prompt to start the program. The initial screen gives the model name and version number. Several information screens with recent developments and other news items then follow. The Main Menu gives the user access to all of the menus described in this chapter.

While the LEAD99d.EXE file occupies only about 160 KB on the hard drive, it will expand to a much larger size when loaded into RAM. Normally, a PC with 640 KB has enough RAM to run the program, but there may be some conflicts with TSR (Terminate and Stay Resident) programs. It may be necessary for the user to remove some TSR programs in order to run the IEUBK Model.

The Model does not require a math co-processor, but calculations may take up to 20 times longer without a co-processor.

2.4.2 Running the Model

The user should fill in the worksheet in Figure 2-11, which defines the exposure scenario, before proceeding with the parameter entries and the computations.

2.4.2.1 Computation Options

The computation menus present the user with a set of computation choices. One choice is the iteration time step, Selection "2". These choices range from 15 minutes to 30 days. The default of 4 hours is adequate for most purposes. Setting this option on the RUN Menu also sets the iteration time for other computation modes, including the Batch Mode.

2.4.2.2 Output Options

At any time during the session, the program may be saved to a designated file. This gives the user the option of retrieving a set of parameters at some future session without reentering the parameters individually. After each model run, the user can select one of several plot options, which can be viewed on the screen, printed to file or sent to a printer. Most plots generated by the model can be printed by using the F10 key on the keyboard. The program presently interfaces with nine standard printer types or orientations. The Graphics Menu selection "7" allows user-specified scaling of the X-axis variable. Future versions of the model may have additional output options.

IEUBK MODEL WORKSHEET			
SITE OR PROJECT:	Model Version:	Date:	
Model Run Control Number:	Site Description:		
PARAMETER	DEFAULT VALUE	USER SELECTED OPTION	UNITS
AIR (constant)			
Outdoor air lead concentration	0.10		$\mu g/m^3$
Ratio of indoor to outdoor air lead concentration	30		%
AIR (by year)			
Air concentration Age = 0-1 year (0-11 mo), 1-2 years (12-23 mo) 2-3 years (24-35 mo) 3-4 years (36-47 mo) 4-5 years (48-59 mo) 5-6 years (60-71 mo) 6-7 years (72-84 mo)	 .10 .10 .10 .10 .10 .10 .10		$\mu g/m^3$
Time outdoors Age = 0-1 year (0-11 mo), 1-2 years (12-23 mo) 2-3 years (24-35 mo) 3-7 years (36-83 mo)	 1 2 3 4		h/day
Ventilation rate Age = 0-1 year (0-11 mo), 1-2 years (12-23 mo) 2-3 years (24-35 mo) 3-4 years (36-47 mo) 4-5 years (48-59 mo) 5-6 years (60-71 mo) 6-7 years (72-84 mo)	 2 3 5 5 5 7 7		m^2/day
Lung absorption	32		%
DATA ENTRY FOR DIET (by year)			
Dietary lead intake Age = 0-1 year (0-11 mo), 1-2 years (12-23 mo) 2-3 years (24-35 mo) 3-4 years (36-47 mo) 4-5 years (48-59 mo) 5-6 years (60-71 mo) 6-7 years (72-84 mo)	 5.53 5.78 6.49 6.24 6.01 6.34 7.00		μg Pb /day

Figure 2-11. Integrated exposure uptake biokinetic model sample worksheet.

DATA ENTRY FOR ALTERNATE DIET SOURCES (by food class)			
Concentration:			µg Pb/g
home-grown fruits	0		
home-grown vegetables	0		
fish from fishing	0		
game animals from hunting	0		
Percent of food class:			%
home-grown fruits	0		
home-grown vegetables	0		
fish from fishing	0		
game animals from hunting	0		
DATA ENTRY FOR DRINKING WATER			
Lead concentration in drinking water	4		µg/L
Ingestion rate			liters/day
Age = 0-1 year (0-11 mo),	0.20		
1-2 years (12-23 mo)	0.50		
2-3 years (24-35 mo)	0.52		
3-4 years (36-47 mo)	0.53		
4-5 years (48-59 mo)	0.55		
5-6 years (60-71 mo)	0.58		
6-7 years (72-84 mo)	0.59		
DATA ENTRY FOR ALTERNATE DRINKING WATER SOURCES			
Concentration			µg/L
first-draw water	4		
flushed water	1		
fountain water	10		
Percentage of total intake			%
first-draw water	50		
flushed water (not a user entry; calculated based on entries for first-draw and fountain percentages)	100 minus first draw and fountain		
fountain water	15		
DATA ENTRY FOR SOIL/DUST (constant)			
Concentration			µg/g
Soil	200		
Dust	200		
Soil ingestion as percent of total soil and dust ingestion	45		%

Figure 2-11 (cont'd). Integrated exposure uptake biokinetic model sample worksheet.

DATA ENTRY FOR SOIL/DUST INGESTION (by year)			
Soil/dust ingestion			
Age = 0-1 year (0-11 mo),	0.085		g/day
1-2 years (12-23 mo)	0.135		
2-3 years (24-35 mo)	0.135		
3-4 years (36-47 mo)	0.135		
4-5 years (48-59 mo)	0.100		
5-6 years (60-71 mo)	0.090		
6-7 years (72-84 mo)	0.085		
DATA ENTRY FOR SOIL (by year)			
Soil lead concentration			µg/g
Age = 0-1 year (0-11 mo)	0		
1-2 years (12-23 mo)	0		
2-3 years (24-35 mo)	0		
3-4 years (36-47 mo)	0		
4-5 years (48-59 mo)	0		
5-6 years (60-71 mo)	0		
6-7 years (72-84 mo)	0		
DATA ENTRY FOR DUST (by year)			
Dust lead concentration			µg/g
Age = 0-1 year (0-11 mo)	0		
1-2 years (12-23 mo)	0		
2-3 years (24-35 mo)	0		
3-4 years (36-47 mo)	0		
4-5 years (48-59 mo)	0		
5-6 years (60-71 mo)	0		
6-7 years (72-84 mo)	0		
DATA ENTRY FOR SOIL/DUST MULTIPLE SOURCE ANALYSIS (constant)			
Ratio of dust lead concentration to soil lead concentration	0.70		unitless
Ratio of dust lead concentration to outdoor air lead concentration	100		µg Pb/g dust per µg Pb/m³ air
DATA ENTRY FOR SOIL/DUST MULTIPLE SOURCE ANALYSIS WITH ALTERNATIVE HOUSEHOLD DUST LEAD SOURCES (constant)			
Concentration			µg/g
household dust (calculated)	150		
secondary occupational dust	1,200		
school dust	200		
daycare center dust	200		
second home	200		
interior lead-based paint	1,200		

Figure 2-11 (cont'd). Integrated exposure uptake biokinetic model sample worksheet.

Percentage household dust (calculated) secondary occupational dust school dust daycare center dust second home interior lead-based paint	 100 minus all other 0 0 0 0 0		%
BIOAVAILABILITY DATA ENTRY FOR ALL GUT ABSORPTION PATHWAYS			
Total lead absorption (at low intake) diet drinking water soil dust alternate source	 50 50 30 30 0		%
Fraction of lead absorbed passively at high intake diet drinking water soil dust alternate source	 0.2 0.2 0.2 0.2 0.2		unitless
DATA ENTRY FOR ALTERNATE SOURCES (by year)			
Total lead intake Age = 0-1 year (0-11 mo), 1-2 years (12-23 mo) 2-3 years (24-35 mo) 3-4 years (36-47 mo) 4-5 years (48-59 mo) 5-6 years (60-71 mo) 6-7 years (72-84 mo)	 0 0 0 0 0 0 0		µg/day
DATA ENTRY MENU FOR MATERNAL-TO-NEWBORN LEAD EXPOSURE			
Mother's blood lead level at time of birth	2.5		µg/dL
DATA ENTRY MENU FOR PLOTTING AND RISK ESTIMATION			
Geometric standard deviation for blood lead, GSD	1.6		unitless
Blood lead level of concern, or cutoff	10		µg/dL
COMPUTATION OPTIONS			
Iteration time step for numerical integration	4		h

Figure 2-11 (cont'd). Integrated exposure uptake biokinetic model sample worksheet.

3. QUICK REFERENCE FOR THE EXPERIENCED USER

3.1 FINDING YOUR WAY THROUGH THE MENUS

The Lead Model is a menu driven program. There is no need to remember special commands, just a cursory understanding of the menu structure. Often you can find what you want just by exploring the various menu options, and familiarity with the model is the best way to shorten this journey. For your reference, a complete menu tree is given in Figure 3-1. Use caution with unfamiliar menu options. Detailed explanations are given in Chapter 2, and more complete documentation may be found in Chapter 4 for most menu options. These should be reviewed before final decisions are made on critical model runs.

3.2 PARAMETER LIST WITH DEFAULT VALUES

The values in Table 3-1 have been assumed for the parameters of the model. These are our best estimates for urban residents with no unusual lead exposures. The estimated blood lead levels with the default parameters represent our best estimate of the blood lead "background" levels that cannot be avoided. The adjustable parameters are listed by screen in the order in which they appear in the model.

Default values are provided for the convenience of the user, but these values may not be appropriate for specific applications. The user has the ultimate responsibility for justification of values used in the applications of the model. We recommend that the user review each of the parameter values in Table 3-1. Most of the parameters will not need to be modified, but the user should be aware of them. Sensitivity analyses on parameters will be useful in documenting results. Many default parameters in the model have only a minor effect on the results (i.e., a 10% change in the air lead concentration parameter will change blood lead levels by less than 1%), but some parameters may be more influential.

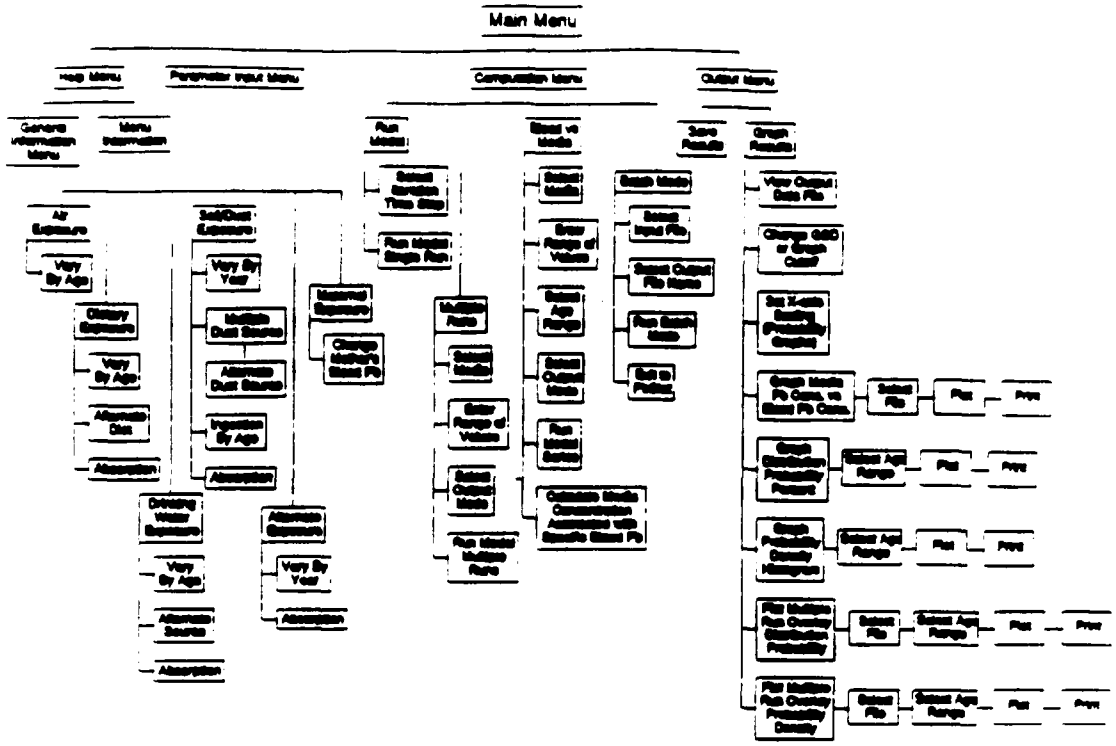


Figure 3-1. Lead model menu tree.

3.3 BATCH MODE INPUT FORMAT

You may find a number of circumstances in which it is convenient to enter input data for many similar exposure scenarios in a single run of the model. Option 4 of the Computation Menu allows you to use a different age or a different value of the lead concentrations in soil, dust, water, air, and alternate lead intake sources for each exposure scenario. The media intake and absorption parameters are the same for every exposure scenario in the run and must be specified before using this option, unless default values are used. These situations may include, but are not limited to:

TABLE 3-1. DEFAULT VALUES FOR MODEL PARAMETERS

PARAMETER	DEFAULT VALUE	UNITS
AIR (constant)		
Outdoor air lead concentration	0.10	$\mu g/m^3$
Ratio of indoor to outdoor air lead concentration	30	%
AIR (by year)		
Air concentration		
Age = 0-1 year (0-11 mo),	.10	$\mu g/m^3$
1-2 years (12-23 mo)	.10	
2-3 years (24-35 mo)	.10	
3-4 years (36-47 mo)	.10	
4-5 years (48-59 mo)	.10	
5-6 years (60-71 mo)	.10	
6-7 years (72-84 mo)	.10	
Time outdoors		
Age = 0-1 year (0-11 mo),	1	h/day
1-2 years (12-23 mo)	2	
2-3 years (24-35 mo)	3	
3-7 years (36-83 mo)	4	
Ventilation rate		
Age = 0-1 year (0-11 mo),	2	m^3 day
1-2 years (12-23 mo)	3	
2-3 years (24-35 mo)	5	
3-4 years (36-47 mo)	5	
4-5 years (48-59 mo)	5	
5-6 years (60-71 mo)	7	
6-7 years (72-84 mo)	7	
Lung absorption	32	%
DATA ENTRY FOR DIET (by year)		
Dietary lead intake		
Age = 0-1 year (0-11 mo),	5.53	μg Pb /day
1-2 years (12-23 mo)	5.78	
2-3 years (24-35 mo)	6.49	
3-4 years (36-47 mo)	6.24	
5-6 years (48-59 mo)	6.01	
5-6 years (60-71 mo)	6.34	
6-7 years (72-84 mo)	7.00	
DATA ENTRY FOR ALTERNATE DIET SOURCES (by food class)		
Concentration:		
home-grown fruits	0	μg Pb/g
home-grown vegetables	0	
fish from fishing	0	
game animals from hunting	0	

TABLE 3-1 (cont'd). DEFAULT VALUES FOR MODEL PARAMETERS

PARAMETER	DEFAULT VALUE	UNITS
Percent of food class:		
home-grown fruits	0	%
home-grown vegetables	0	
fish from fishing	0	
game animals from hunting	0	
DATA ENTRY FOR DRINKING WATER		
Lead concentration in drinking water	4	µg/L
Ingestion rate		
Age = 0-1 year (0-11 mo),	0.20	liters/day
1-2 years (12-23 mo)	0.50	
2-3 years (24-35 mo)	0.52	
3-4 years (36-47 mo)	0.53	
4-5 years (48-59 mo)	0.55	
5-6 years (60-71 mo)	0.58	
6-7 years (72-84 mo)	0.59	
DATA ENTRY FOR ALTERNATE DRINKING WATER SOURCES		
Concentration		µg/L
first-draw water	4	
flushed water	1	
fountain water	10	
Percentage of total intake		
first-draw water	50	%
flushed water	100 minus first draw and fountain	
fountain water	15	
DATA ENTRY FOR SOIL/DUST (constant)		
Concentration		
soil	200	µg/g
dust	200	
Soil ingestion as percent of total soil and dust ingestion	45	%
DATA ENTRY FOR SOIL/DUST INGESTION (by year)		
Soil/dust ingestion		
Age = 0-1 year (0-11 mo),	0.085	g/day
1-2 years (12-23 mo)	0.135	
2-3 years (24-35 mo)	0.135	
3-4 years (36-47 mo)	0.135	
4-5 years (48-59 mo)	0.100	
5-6 years (60-71 mo)	0.090	
6-7 years (72-84 mo)	0.085	

TABLE 3-1 (cont'd). DEFAULT VALUES FOR MODEL PARAMETERS

PARAMETER	DEFAULT VALUE	UNITS
DATA ENTRY FOR DUST (by year)		
Dust lead concentration		
1-2 years (12-23 mo)	0	µg/g
2-3 years (24-35 mo)	0	
3-4 years (36-47 mo)	0	
4-5 years (48-59 mo)	0	
5-6 years (60-71 mo)	0	
6-7 years (72-84 mo)	0	
DATA ENTRY FOR SOIL/DUST MULTIPLE SOURCE ANALYSIS (constant)		
Ratio of dust lead concentration to soil lead concentration	0.70	unitless
Ratio of dust lead concentration to outdoor air lead concentration	100	µg Pb/g dust per µg Pb/m^3 air
DATA ENTRY FOR SOIL/DUST MULTIPLE SOURCE ANALYSIS WITH ALTERNATIVE HOUSEHOLD DUST LEAD SOURCES (constant)		
Concentration		
household dust	150	µg/g
secondary occupational dust	1,200	
school dust	200	
daycare center dust	200	
second home	200	
interior lead-based paint	1,200	
Percentage		
household dust	100 minus all other	%
secondary occupational dust	0	
school dust	0	
daycare center dust	0	
second home	0	
interior lead-based paint	0	
BIOAVAILABILITY DATA ENTRY FOR ALL GUT ABSORPTION PATHWAYS		
Total lead absorption (at low intake)		
diet	50	%
drinking water	50	
soil	30	
dust	30	
alternate source	0	
Fraction of lead absorbed passively at high intake		
diet	0.2	unitless
drinking Water	0.2	
soil	0.2	
dust	0.2	
alternate source	0.2	

TABLE 3-1 (cont'd). DEFAULT VALUES FOR MODEL PARAMETERS

PARAMETER	DEFAULT VALUE	UNITS
DATA ENTRY FOR ALTERNATE SOURCES (by year)		
Total lead intake		
Age = 0-1 year (0-11 mo),	0	µg/day
1-2 years (12-23 mo)	0	
2-3 years (24-35 mo)	0	
3-4 years (36-47 mo)	0	
4-5 years (48-59 mo)	0	
5-6 years (60-71 mo)	0	
6-7 years (72-84 mo)	0	
DATA ENTRY MENU FOR MATERNAL-TO-NEWBORN LEAD EXPOSURE		
Mother's blood lead level at time of birth	2.5	µg/dL
DATA ENTRY MENU FOR PLOTTING AND RISK ESTIMATION		
Geometric standard deviation for blood lead, GSD	1.6	unitless
Blood lead level of concern, or cutoff	10	µg/dL
COMPUTATION OPTIONS		
Iteration time step for numerical integration	4	h

(1) comparison of predicted values from the Integrated Exposure/Uptake Biokinetic (IEUBK) model with actual blood lead levels observed in a blood lead and environmental lead field study such as illustrated in Table 3-2.;

(2) risk estimation using predicted values from the IEUBK model with actual environmental lead levels observed in an environmental lead field study;

(3) estimation of the effect of variability in environmental lead levels on the distribution of blood lead levels in the population;

(4) sensitivity analyses on the impact of environmental lead exposure.

The input data file for a batch run must be created outside the IEUBK model using whatever text editor the user prefers. The following conventions MUST be observed in creating the batch file:

TABLE 3-2. FORMAT FOR BATCH MODE INPUT DATA FILE (SIMULATED DATA)

Header line 1 > INPUT DATA FILE FOR A MONTE CARLO SIMULATION

Header line 2 > FIRST THREE COLUMNS RANDOM NOS. FOR SOIL, DUST, BLOOD

Header line 3 > ID FAM NBHD AGE PBS PBD PBW PBA ALT PBB

1	FP-1	PIONEER_HILL	18	510.0	457.8	4.0	0.1	0.0	2.6
2	FP-2	PIONEER_HILL	18	200.0	159.8	4.0	0.1	0.0	2.3
3	FP-3	PIONEER_HILL	18	373.1	475.3	4.0	0.1	0.0	3.4
4	FP-4	PIONEER_HILL	18	1042.5	1361.6	4.0	0.1	0.0	8.7
5	FP-5	PIONEER_HILL	18	519.2	332.7	4.0	0.1	0.0	2.9
6	FP-6	PIONEER_HILL	18	3123.6	1117.6	4.0	0.1	0.0	6.3
7	FP-7	PIONEER_HILL	18	1938.2	1295.8	4.0	0.1	0.0	4.9
8	FP-8	PIONEER_HILL	18	287.5	649.9	4.0	0.1	0.0	11.8
9	FP-9	PIONEER_HILL	18	1227.0	1997.5	4.0	0.1	0.0	23.3
10	FP-10	PIONEER_HILL	18	321.7	405.5	4.0	0.1	0.0	4.4
11	FR-1	RIVERSIDE	18	631.9	58.4	4.0	0.1	0.0	5.6
12	FR-2	RIVERSIDE	18	55.7	23.9	4.0	0.1	0.0	2.0
13	FR-3	RIVERSIDE	18	336.3	658.6	4.0	0.1	0.0	4.1
14	FR-4	RIVERSIDE	18	666.9	221.5	4.0	0.1	0.0	3.2
15	FR-5	RIVERSIDE	18	2005.6	1532.1	4.0	0.1	0.0	26.2
16	FR-6	RIVERSIDE	18	394.8	92.7	4.0	0.1	0.0	2.3
17	FR-7	RIVERSIDE	18	1183.3	692.8	4.0	0.1	0.0	2.7
18	FR-8	RIVERSIDE	18	450.8	83.6	4.0	0.1	0.0	2.4
19	FR-9	RIVERSIDE	18	210.2	94.7	4.0	0.1	0.0	1.5
20	FR-10	RIVERSIDE	18	650.6	682.0	4.0	0.1	0.0	4.5
21	FR-11	RIVERSIDE	18	238.4	314.2	4.0	0.1	0.0	2.0
22	FR-12	RIVERSIDE	18	256.6	48.0	4.0	0.1	0.0	2.0
23	FR-13	RIVERSIDE	18	636.2	219.6	4.0	0.1	0.0	4.4
24	FR-14	RIVERSIDE	18	100.3	31.5	4.0	0.1	0.0	3.2
25	FB-1	BRIDGE_ST	18	678.6	605.4	4.0	0.1	0.0	10.0
26	FB-2	BRIDGE_ST	18	249.5	136.3	4.0	0.1	0.0	2.0
27	FB-3	BRIDGE_ST	18	291.5	127.8	4.0	0.1	0.0	4.1
28	FB-4	BRIDGE_ST	18	750.9	2514.8	4.0	0.1	0.0	4.5
29	FB-5	BRIDGE_ST	18	89.3	135.4	4.0	0.1	0.0	3.3
30	FB-6	BRIDGE_ST	18	943.2	282.4	4.0	0.1	0.0	6.7
31	FB-7	BRIDGE_ST	18	1668.6	369.2	4.0	0.1	0.0	11.9
32	FB-8	BRIDGE_ST	18	1399.1	509.0	4.0	0.1	0.0	6.0
33	FB-9	BRIDGE_ST	18	1226.8	509.0	4.0	0.1	0.0	5.9
34	FB-10	BRIDGE_ST	18	1905.1	978.3	4.0	0.1	0.0	4.6
35	FB-11	BRIDGE_ST	18	366.3	2727.8	4.0	0.1	0.0	13.4
36	FB-12	BRIDGE_ST	18	62.5	464.7	4.0	0.1	0.0	3.9
37	FB-13	BRIDGE_ST	18	75.8	49.3	4.0	0.1	0.0	5.4
38	FB-14	BRIDGE_ST	18	393.5	88.6	4.0	0.1	0.0	1.2
39	FB-15	BRIDGE_ST	18	461.6	883.7	4.0	0.1	0.0	1.7
40	FB-15	BRIDGE_ST	18	461.6	883.7	4.0	0.1	0.0	1.9

- The input data file must be an ASCII file with no special characters.
- The data set must have a DAT extension (i.e., [name].DAT).
- The first three lines of the input data file can be any identifiers that the user requires; we usually use the first line for the run name, the second line for modelling options used in the run, and the third line as headers for variables in the data set;
- The data fields are entered format-free, although the use of regular spacings and alignment of decimal points are recommended to improve readability;
- Maximum width 80 columns;
- Variable values should be separated by spaces;
- Missing values must be shown by an isolated decimal point as in some examples in Chapter 5;
- Each line in the input data file must contain the following 10 variables:

1. Child identifier or code
2. Family or residence unit identifier or code
3. Area or neighborhood identifier code
4. Child's age in months
5. Soil lead concentration in μg Pb/g
6. Dust lead concentration in μg/g
7. Drinking water lead concentration in μg/L
8. Air lead concentration in $\mu g/m^3$
9. Daily intake of lead from other sources, μg Pb/day
10. Observed child blood lead level.

In Chapter 5 we will demonstrate an approach to using the IEUBK model in the batch mode option. Soil, dust, and blood lead "data" were simulated using realistic parameters for sites with active air lead point sources. The attached Table 3-2 shows a batch mode input data file for 40 children. All were assumed to be 18 months old and had default air, water, and alternate source lead values. The first three columns in Table 3-2 are the child identifier, the family identifier (note simulated twins as ID 39 and 40), and the "neighborhood". These fields could be any alpha-numeric identifiers defined by the user.

3.4 OUTPUTS FOR DOCUMENTATION, BRIEFING, AND PRESENTATION

3.4.1 Overview of Output Options

The IEUBK model includes some output options that facilitate presentation of the results and testing of the results for parameter sensitivity. These options may be a useful part of the documentation for decisions in which the IEUBK model plays a role. We will first describe the output options, and then show some of their applications. Some sensitivity analyses can be facilitated by the use of these options.

3.4.1.1 Plotting (Option 2)

Single-Plot Options (Selections 2 and 3)

Some plot options can be used with single runs of the IEUBK model, and others require multiple runs. Single-run options include:

- Plotting the log-normal probability density function of blood lead levels for a single exposure scenario predicting geometric mean blood lead (Selection 3);
- Plotting the cumulative probability distribution for exceeding any user specified blood lead level of concern for a single exposure scenario (Selection 2). This is sometimes called the exceedance probability distribution.

The probability density function gives most users a better idea of the spread of blood lead levels for children exposed to a single set of environmental lead concentrations. The exceedance probability distribution may be used to visually estimate the fraction of children above a blood lead level of concern for the single-exposure case (e.g., what fraction of children are above 10 μg/dL), or to visually estimate the blood lead level corresponding to a specified fraction of children (e.g., what blood lead concentration encompasses 95% of the children). The user may route the probability plots to a printer after viewing the display.

Multiple-Plot Options (Selections 1, 4, and 5)

There are additional features that allow the user to combine output from several runs onto single plots. These multiple-run options include:

- Overlaid plots of the log-normal probability density functions of blood lead levels for multiple exposure scenarios, where each run increases the lead concentration in a specified medium by a user-defined amount (Selection 5);

- Overlaid plots of the cumulative exceedance probability distributions of blood lead levels for multiple exposure scenarios, where each run increases the lead concentration in a specified medium by a user-defined amount (Selection 4);

- Plots of geometric mean blood lead levels versus environmental lead levels for the medium whose values are varied (Selection 1).

Selection 1 cannot be used unless the user has previously created an output file from the Computation Menu, designated *.PBM. Selections 4 or 5 cannot be used unless the user has previously created an output file from the Computation Menu, designated *.LAY.

The overlaid probability density functions give most users a better idea of how the probability of exceeding a blood lead level of concern increases with each increment in environmental lead. The exceedance probability distributions may be used to estimate the increases in the fraction of elevated blood lead levels or to visually estimate the environmental lead levels corresponding to a specified fraction of non-protected children above the level of concern.

3.4.1.2 Uses of Batch Mode Analysis (Option 4)

Results from multiple exposure scenarios can be accumulated using the batch mode options. For sensitivity analyses involving constant concentrations in air, water, dust, soil, or an alternate medium whose intake is constant, it is possible to create a batch mode input file in which each line represents a different case for the analysis. However, with Option 4 it is only possible to carry out sensitivity analyses in which the cases differ on the basis of concentration. Other types of sensitivity analyses require the accumulation of single runs in an overlay file.

Another application in which batch mode methods are useful is a Monte Carlo analysis in which all the modelled variability is assigned to differences in the environmental concentrations. The results can be stored in a batch mode output file which may then be used for statistical analyses.

3.4.2 Detailed Instructions on Output Options

3.4.2.1 Save Output from a Single Run

1. Develop an exposure scenario.

2. Run the model (Selection 2 on Computation Menu, or F5 from any Data Entry Menu).

3. Save results (Selection 2 after running the model). Results may be appended to the file RESULTS.TXT, or added to an overlay plot file defined by the user with name [name].LAY.

4. Results may be sent to a printer.

3.4.2.2 Save Output from Multiple Runs for Probability Plots (Option 3 on Range Selection Menu)

1. Develop an exposure scenario

2. Use the Multiple Runs option (Option 2) on the Computation Menu.
   1- Select the medium (Soil, Dust, Air, Water, Diet)
   2- Select the lower and upper values for the medium
   4- OUTPUT CHOICES
      - Select number of steps from small to large
      - Send results to overlay file RANGE(#+1).LAY, where the output file is automatically named by increasing the index of the largest numbered (#) current RANGE#.LAY file.

3. Results may be sent to a printer.

3.4.2.3 Save Output from Multiple Runs for Media-Level Plots (Option 3 on the Computation Menu)

1. Develop an exposure scenario

2. Use the Media Run option (Option 3) on the Computation Menu.
   1- Select the medium (Soil, Dust, Air, Water, Diet)
   2- Select the lower and upper values for the medium
   4- OUTPUT CHOICES
      - Select number of steps from small to large
      - Change the age range for calculating mean blood lead.
      - Send results to overlay file MEDBLD(#+1).PBM, where the output file is automatically named by increasing the index of the largest current MEDBLD#.PBM file.

3. Results may be sent to a printer.

3.4.2.4 Save Output from a Batch Mode Run (Option 4 on the Computation Menu)

1. Create a batch mode input data file, [name].DAT.
2. Use the Batch Mode Run option (Option 4) on the Computation Menu.
3. Load the [name].DAT file you have created.
4. Rename the output data file [newname].* if required in the Run step.
5. Run the model.

The output data sets are named [newname].ASC and [newname].TXT, or [name].ASC and [name].TXT if not renamed.

3.4.2.5 Probability Plots for Single Runs

For Current Runs Using Option 2 on Output Menu:

1. Run the model with user-defined exposure scenario in Option 2 on Output Menu.
2. Then choose Selection 2 or 3 in the Graphics Selection Menu.
3. Choose the Age Range.
4. Print graph, without exiting, by using the F10 key, then selecting printer type.

If the user has exited from the current Run, but has not done any further runs, then Steps 3 through 5 can be executed by returning to the Graphics Menu and executing Option 2 or 3.

For Current Runs Using Option 2 on Output Menu:

1. Run the model with user-defined exposure scenario in Option 2. If the user has aborted the runs in Option 2, but has not done any runs since, then the last complete run may be plotted as above. The procedure is:
2. Select Option 3 in the Main Menu, then option 2 on the Output Menu.
3. Then choose Selection 2 or 3 in the Graphics Selection Menu.
4. Choose the Age Range.
5. Print graph, without exiting, by using the F10 key, then selecting printer type.

3.4.2.6 Probability Plots for Multiple Runs

1. Run the model with user-defined exposure scenario in Option 2 on the Computation Menu. Do not abort the runs in Option 2.
2. Select Option 3 in the Main Menu, then Option 2 on the Output Menu.
3. Then choose Selection 4 or 5 in the Graphics Selection Menu.
4. Identify the *.LAY data set to plot.
5. Choose the Age Range.
6. Print graph, without exiting, by using the F10 key, then selecting printer type.

3.4.2.7 Multi-Level Plots for Blood Lead Versus Media Lead

1. Run the model with user-defined exposure scenario in Option 2 on the Computation Menu.
2. Select Option 3 in the Main Menu, then Option 2 on the Output Menu.
3. Then choose Selection 1 in the Graphics Selection Menu.
4. Identify the MEDBLD#.PBM data set to plot.
5. Print graph, without exiting, by using the F10 key, then selecting printer type.

Batch mode files can be used for display or documentation through the statistics module on the Batch Mode Menu, Option 4.

3.4.3 Recommendations on Multi-Level Soil Lead Exposure Scenarios

The IEUBK model carries out multi-level analyses by increasing the concentration of the user-specified medium by equal steps at each run, and holding everything else consta· When evaluating different soil lead exposure scenarios, it may be preferable to keep a constant soil-to-dust coefficient so that dust lead exposure increases with increasing soil lead exposure. This can be done by first invoking the Multiple Source Analysis for dust and defining the dust lead to soil lead coefficient. This is particularly important if some component of the soil lead abatement is expected to permanently alter the soil to dust pathway.

4. MORE ABOUT THE MODEL[1]

4.1 LEAD BIOAVAILABILITY

4.1.1 Background

The concept of bioavailability is important for site-specific risk assessments for lead. The concept springs from the fact that lead potentially available to produce harm and found in exposure pathways or in body receiving compartments (lung, skin, gut) must reach the biological sites of action in order for an adverse health effect to occur in exposed humans or ecological biota.

This section focuses primarily on the bioavailability of inorganic lead from soils and dusts. Lead bioavailability from air and drinking water is also important and is discussed in limited detail below. In order to provide coherent and useful guidance to the reader and user of this chapter, it is subdivided into (1) introductory material that includes definitions of bioavailability and resource material in the technical literature; (2) the close lead absorption-bioavailability relationships, including the physiological and biochemical mechanisms of lead absorption and the many, complex factors that influence such uptake; (3) the main focus of the chapter, bioavailability as it relates to human and experimental toxicology, including the various biophysico-chemical and environmental aspects of the lead exposure matrix, methodological approaches in toxicology for quantifying bioavailability, the increasingly important question of relevant experimental animal models for quantifying lead bioavailability in humans; and, finally, (4) a summary and critical overview, which attempts to spell out the appropriate uses of bioavailability information and limits to use this information in site-specific risk assessment.

4.1.2 Definitions

A clear agreement on a definition of bioavailability should be established before one presents a detailed discussion of this topic. The difficulty here is that there are various

[1]This chapter is intended to provide guidance on some technically advanced applications of the model. We have attempted to provide the best scientific documentation available but recognize that new information may become available in these rapidly advancing fields. The user is referred to Section 1.6 for information on how to get additional and more up-to-date assistance with specific applications of the model.

definitions of bioavailability depending on the scientific discipline using the term and the technical context of use.

Typically, the pharmacologist or toxicologist or others in biomedical disciplines are concerned with measuring bioavailability as that fraction of the total amount of material in contact with a body portal of entry (lung, gut, skin) that then enters the blood. For the purpose of describing the Integrated Exposure Uptake Biokinetic (IEUBK) Model, this is the definition to be used in this manual. However, an aquatic biologist may define bioavailability as that fraction of material solubilized in the water column under certain conditions of hardness and pH. An aquatic toxicologist might consider contaminants which are soluble under specific stream conditions to be bioavailable to fish or benthic organisms. A biochemist or biochemical toxicologist would consider bioavailability with reference to that fraction of a toxicant which is available at the organ or cellular site of toxicity.

The above definitions can be viewed as dosimetrically descriptive. There are quantitative methodological definitions that figure as well. As described later, bioavailability can be defined as being absolute or relative (comparative). Absolute bioavailability, for example, is the amount of substance entering the blood via a particular biological pathway relative to the absolute amount that has been ingested. Relative bioavailability of lead is indexed by comparing the bioavailability of one chemical species or form of lead with that of another form of lead. A second methodological description for bioavailability that is used by toxicologists is the ratio of areas under the dose-response curve for either of two forms of lead, or two methods of administration. Typically, the latter involves comparing injected with orally administered doses.

4.1.3 Literature Sources on Bioavailability

More detailed reviews and discussions of the topic of lead bioavailability in humans and experimental animals have been presented by Mushak (1991) and Chaney et al. (1988). As is evident from these reviews, our present understanding of lead bioavailability has developed from both human and animal studies. For further in-depth discussion of the various components of bioavailability, for example, lead absorption, the reader is also referred to the following documents: (1) the Air Quality Criteria Document for Lead (U.S. Environmental Protection Agency, 1986), and (2) the Proceedings of the Symposium on the Bioavailability and Dietary Exposure of Lead (1991).

Citations of key specific studies are provided in the relevant sections and subsections of this chapter rather than here, so as to be less disruptive to the reader.

4.1.4 Lead Absorption-Bioavailability Relationships

By definition, the absorption (uptake) of lead into the circulation is the critical kinetic component of the overall process called bioavailability. Not only the amount, but also the *rate of uptake* of that given amount is important, particularly under acute or subacute exposure conditions, and when dealing with lead-containing media in the gastrointestinal (GI) tract. Such material is itself moving through the GI tract within a relatively short time period. Consequently, the biological and physiological characteristics of absorption, the subcellular mechanisms of absorption, and the factors influencing its occurrence must be understood in order to understand the resulting phenomenon. The focus of this chapter is soil and dust lead ingested (swallowed) by populations at risk, requiring that lead uptake phenomena in the gastrointestinal tract be given most of the attention.

Species-specific anatomical and physiological determinants of GI absorption are the macroscopic factors that provide the basic means by which lead absorption occurs. As noted in more detail in Section 4.1.5, there are major structural differences in the anatomy of the GI tract of various mammalian species that would affect lead absorption. Similarly, it is the physiology of the mucosal lining (epithelium) of the mammalian GI tract that is the first dynamic determinant of lead movement from the GI tract to the bloodstream.

4.1.5 Cellular and Subcellular Mechanisms of Lead Absorption

Lead absorption is believed to proceed by several cellular mechanisms involving the enterocytes, cells lining the intestinal wall (Figure 4-1) (e.g., Mushak, 1991). Absorption also entails complex interactions with the uptake of essential nutrients such as calcium, iron and phosphate (Barton et al., 1978, 1981; Mahaffey-Six and Goyer, 1972).

The first uptake mechanism may be diffusion through the gut lumen driven by a concentration gradient from the luminal surface lining the intestine to the basolateral surface (vascular side). This mechanism is likely to depend to some extent on the concentration of ionic or unbound lead ion (Pb^{2+}), and consequently would depend on the solubility characteristics of lead species of interest. This may be a passive diffusion process requiring no energy input. It involves either intracellular or paracellular movement of lead across the

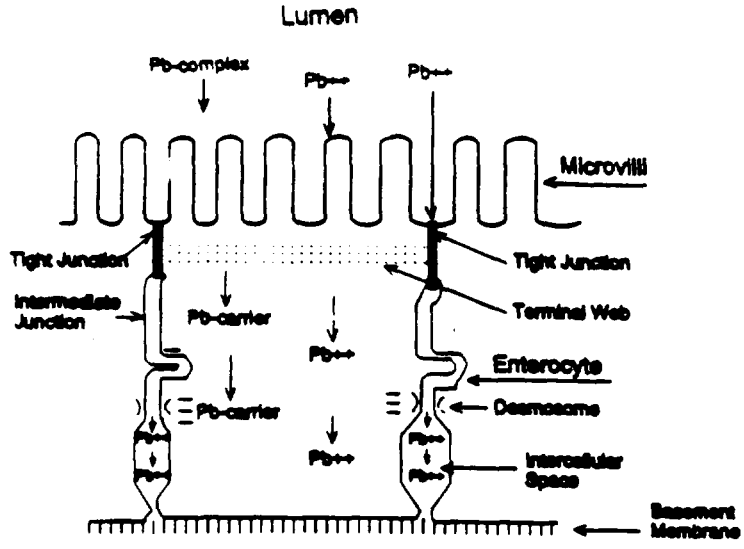


Figure 4-1. Schematic drawing of the enterocyte showing possible mechanisms for lead absorption. Possible mechanisms include: (1) an active or facilitated component; (2) a transcellular component perhaps involving pinocytotic mechanisms; and (3) a diffusion-driven paracellular route across tight junctions.

Source: Mushak, 1991, adapted from Morton et al. (1985).

wall. Paracellular transport would entail movement across the area between cells called "tight junctions."

In the second possibility, lead may enter the gut tissue (but not necessarily the bloodstream) by pinocytosis or other vesicular mechanisms. In pinocytosis, lead-bearing media in a liquid micro region of the gut are engulfed by the (enterocyte) cell membrane. Such encapsulating may involve lead in either a truly soluble or an emulsified/suspended form that is then carried to blood or to sites of toxic action. This process is biochemically analogous to handling of solid particles in phagocytosis.

Perhaps the quantitatively most important transport mechanism in environmental exposures typical for most individuals is energy-driven active transport, exploiting homeostatic transport mechanisms in place for calcium and iron transport (e.g., calcium binding protein [CaBP] or calbindin D), and under control of an enzyme—calcium, magnesium-dependent ATPase (Ca^{2+}, Mg^{2+}-ATPase)—involved in the absorption and regulation of blood calcium levels and located in the basolateral membrane of mucosal epithelial cells. This active component of lead absorption displays a strong age dependence,

being more important at younger ages. It is interesting that some of the transport systems that bring calcium into the body seem to have an even higher affinity for lead than for calcium (e.g., Fullmer et al., 1985).

While the results of experimental studies can be described quantitatively, the precise nature of biological and biochemical mechanisms in lead bioavailability is not yet completely understood. There is, however, a useful characterization of lead absorption mechanisms as either saturable (facilitated) or nonsaturable (passive). These various and complex biochemical/cellular mechanisms obviously have important implications for experimental models of human lead bioavailability, particularly with reference to comparison of in vivo to in vitro simple chemical simulation models.

4.1.6 Factors Affecting Lead Absorption

Lead uptake, especially from the GI tract, does not occur in a physiological vacuum but is the outcome of a complex set of interactions with other inorganic and organic substances, particularly such nutrients as calcium, iron, phosphate, vitamin D, fats, etc., as they occur in meals or with intermittent eating. In addition, uptake is a function of developmental stage (age), administered dose, the chemical species and the particle size of the lead-containing media.

It is well known that lead uptake is markedly lower with consumption of meals than under fasting conditions in adults (e.g., James et al., 1985; Rabinowitz et al., 1980) and presumably in children as well. Human data, in the aggregate, indicate that calcium, iron and other cations interact strongly as competitors to lead uptake so that lead uptake generally increases as dietary levels of these nutrients decrease (Mushak, 1991; U.S. Environmental Protection Agency, 1986). In rats, Garber and Wei (1974) showed that fasting increased the amount of lead taken up by the gut. Children are likely to be exposed to lead under a variety of fed or fasted (between meal) conditions. Therefore, any interpretations of lead bioavailability studies of site-specific characteristics should include the effect on uptake of food and time since eating.

There is a developmental or age dependency for the extent of lead absorption in both humans and experimental animals (Mushak, 1991; U.S. Environmental Protection Agency, 1986). Prepubertal children absorb more lead than do adults (Alexander et al., 1973; Ziegler et al., 1978). Experimental animal studies support the human data. Studies using rats showed that pre-weanling animals absorb 40 to 50 times more of a given dose of lead than

do adult animals (Kostial et al., 1971, 1978; Forbes and Reina, 1972), while infant monkeys will absorb 16 to 21 times more lead than adult monkeys (Munro et al., 1975). Possible mechanisms for this age dependence have been discussed (Weis and LaVelle, 1991; Mushak, 1991). The design or interpretation of bioavailability studies, aimed at assessing lead absorption for children, must consider age dependence of uptake of lead in any adjustments of the bioavailability parameter in the UBK model.

Human data indicate a dose dependence to the absorption of lead (Sherlock and Quinn, 1986). In duplicate diet studies of bottle-fed infants (5 to 7 kg) exposed to lead in water and in formula mixed with contaminated water, Sherlock and Quinn were able to quantify the dose dependence of lead absorption. Over the exposure range investigated in the study (40 to 3,000 μg/week), these investigators determined that the relationship between blood lead concentration and lead intake was curvilinear (Figure 4-2). This opportunistic human data describing the dose-dependence of lead absorption was considered by the Agency when establishing the kinetic approach to lead absorption used in the IEUBK Model.

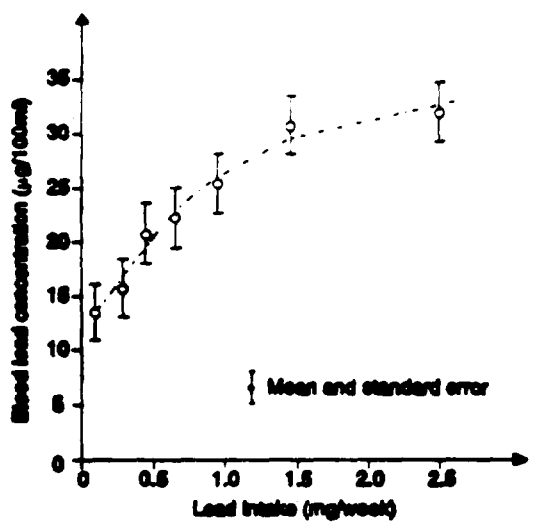


Figure 4-2. Dose-dependent relationship between dietary lead (formula mixed with water) and blood lead in infants.

Source: Sherlock and Quinn (1986).

Animal studies (e.g., Bushnell and DeLuca, 1983) indicate that GI lead absorption shows dependence on the level of oral dosing. Bushnell and DeLuca reported that lead uptake rates decreased when oral lead exposure concentration exceeded 10 to 100 ppm. This

dose-dependent inhibition of uptake is consistent with an active transport mechanism that requires lead-inhibited enzyme(s) for its operation and which also becomes saturated at higher lead dosings (Aungst and Fung, 1981; Mykkanen and Wasserman, 1981). Design and interpretation of studies to assess bioavailability of lead should also consider dose dependency in site-specific assessments.

Finally, the metal species and particle size may influence the solubility, and because of that, the bioavailability of lead. Experimental studies using relatively simple lead species showed that lead as the sulfide, chromate, naphthenate or octoate was less bioavailable (44 to 67%) relative to the more soluble carbonate (Baritrop and Meek, 1975). Baritrop and Meek (1979) also demonstrated an inverse relationship between lead uptake from leaded paint and particle size.

On the other hand, other investigators have documented that lead species that are relatively insoluble under simple in vitro conditions are as bioavailable as soluble salts under conditions of fasting (LaVelle et al., 1991; Rabinowitz et al., 1980).

4.1.7 Bioavailability of Lead in Soils and Dusts

Quantitative approaches to estimating bioavailability for purposes of the IEUBK model require consideration of three issues. The first, of course, is the physicochemical nature of the site-specific environmental media containing lead and what this suggests for behavior of lead-containing media in the GI tract (i.e., biophysico-chemical behavior). As noted earlier, particle size and chemical species are important. Equally important is the environmental matrix within which some particular chemical species of lead is to be found. The physicochemical complexity of these environmental matrices (e.g., dusts and soils, mining and process waste) considerably exceeds that of simple, laboratory forms. The second aspect is methodological: how one can quantify bioavailability in experimental or observational studies? Finally, it is critical that users of this manual and model understand the merits and the limits of the various types (classes) of bioavailability studies that can be done on a site-specific basis.

4.1.7.1 Biophysico-Chemical and Environmental Features of the Exposure Matrix

Types of Soil Lead Contamination

Environmental lead is found in a variety of chemical and physical forms. Lead-contaminated areas could be categorized according to the type of industry or lead-generating

processes associated with the site. Since we are concerned principally with lead in dusts and soils, these are the media of most site-specific concern.

Urban area sites are typically contaminated with those chemical forms arising from either the combustion of leaded gasoline (alkyl lead species such as tetraethyl lead used as anti-knock agents) at high levels in past years or from flakes, chips and dusts from exterior and interior lead-based paint.

Dust or soil lead originating from auto exhaust typically begins as lead-mixed halides (chloride, bromide) but undergoes transformation quickly to the oxide or sulfate (U.S. Environmental Protection Agency, 1986), two relatively bioavailable forms. Auto emission particulate is typically of small diameter (one micron or less), especially on residential surfaces farther away from roadways, where distant atmospheric transport is more favored than for the heavier particles that are deposited closer to the traffic sources. Such particles are also readily breathed into the lungs and readily stick to the hands of children, to family pets, etc. (U.S. Environmental Protection Agency, 1986; Mushak, 1991).

Paint lead is typically found as carbonate, chromate or octoate, and the element may represent up to 70% of the weight of the dried paint product. While lead-paint surfaces are intact, leaded paint would only become available as young children chew on accessible surfaces like painted furniture. In older structures, with surface aging, window and door frame abrasion, and deterioration of leaded paint surfaces, paint will flake, chip, chalk (interior) or weather (exterior) and become an important source of lead exposure for children. The nature of this material, especially as small adherent flecks and fine dusts, and its significant solubility are factors likely to favor significant bioavailability. The greatest numbers of lead-painted residential units are found in urban areas, but any unit anywhere built before 1978 may have lead-based paint.

Battery recycling plants, typically containing secondary lead smelting capacity, are often found as localized sources of environmental lead. Waste byproducts of this kind of lead processing include lead sulfate (sulfuric acid) on casings, and battery sulfuric acid itself, mobilizing lead into and through soils of limited buffering capacity. Lead from this material, either as feedstock or from secondary smelter stack emissions, is apt to be of small particle size as well. These factors warrant estimating bioavailability at the upper end of the range.

In nonferrous mining areas, lead is commonly found in a variety of material produced by hard rock mining, milling, and smelting processes. It is beyond the scope of this chapter

to present a detailed discussion of lead contamination with nonferrous mining, milling and smelting. The reader is referred to the review by Mushak (1991) for further details.

Mining waste can be broadly characterized as: (1) waste rock; (2) mill tailings; and (3) smelting waste. Waste rock is that material removed from the mine but having insufficient mineral economic value to warrant processing. This material is typically discarded at openings to the mine, consists of larger particles, and may or may not be enriched in heavy metals.

Mill tailing is material that has been processed by a variety of physical grinding, separating and enrichment processes. This material typically has smaller particle size than the less processed wastes and the material is enriched in toxic elements, including lead. Mineral content depends on the characteristics of the ore body and the milling process, and may range from soluble carbonates ($K_{sp} \approx 10^{-8}$) to extremely insoluble phosphates ($K_{sp} \approx 10^{-80}$) of lead. Furthermore, lead that is associated with mining waste may either be freely exposed at the particle surface or entirely encapsulated, so that the lead is not available to be dissolved in simple solvents like water.

Smelting waste may exist in many forms. Air- and water-quenched slags are strikingly different in their physical nature. Water-quenched material is typically of fine particle size, while air quenching results in large chunks of oxidized slag. Chemically, these slags consist of various metal oxides and include lead and silicon oxides. Bag house dust consists of the fine particulate matter trapped in the emissions stream by a simple bag filter prior to leaving the stack. This material is very high in toxic metal content, including lead, and occurs in very small particle size. These small particles include lead sulfate and oxide species. Dross is the foam or lighter fraction of the liquid product of the floatation process. When cool, it may be discarded, resulting in a potentially important exposure source.

4.1.7.2 Is There a Better Way To Classify Lead-Contaminated Sites?

It is often convenient to discuss lead-contaminated sites by classifying them as mining, smelting, urban or battery sites. As our understanding of the complexities of lead-contaminated sites improves, it becomes less and less useful to use these simplified descriptions. For example, mining areas typically are associated with present or historical milling and smelting. Significant smelter-related contamination may remain at closed and operating mines that can contribute to typical mine waste exposure concerns.

Mine wastes may consist of lead in a multitude of physical and chemical forms as discussed above, making generalizations about exposure or potential exposure (and bioavailability) inappropriate without additional applied research data. Mining and smelting areas may share exposure sources often associated with such urban areas. Adequate characterization of lead-contaminated media, for the purpose of estimating bioavailability, should include assessment of physical and chemical parameters (e.g., particle size and appropriate media solubility) as well as biophysico-chemical characteristics. Generalizations regarding the source of lead contamination which do not address risk-specific details of the physicochemical and biochemical nature of the waste are not as useful for predicting health risks from exposures.

4.1.7.3 Methodological Approaches to Quantifying Bioavailability

While lead can have severe toxic effects following a single very high exposure, we are primarily concerned in this chapter with relatively low levels of average exposure and average blood lead concentration (see Figures 4-3 and 4-4 for single versus multiple exposures and target organ concentrations).

The average near steady state (pseudoequilibrium) of an accumulating toxicant such as lead in blood following chronic (repetitive) exposure is proportional to the amount absorbed during each exposure. At low ingestion rates, where absorption and biokinetic processes are nearly linear, the following relationship applies between changes in blood lead and changes in chronic exposure:

$$\Delta \text{ PbB} = \frac{\Delta \text{ Pb-abs./day} \times \text{mean residence time in blood pool}}{\text{volume of distribution in blood pool}}.$$

Methods used to describe the fraction absorbed from exposure are well established and will be the primary focus of the following discussion.

4.1.7.4 Determination of Absolute Bioavailability

The methodology for quantifying absolute bioavailability in toxicology commonly compares (a) the area under the time-versus-blood-concentration curve (AUC) following intravenous (IV) injection with (b) an equivalent dose and a similar AUC measurement following ingestion of the substance being investigated. The ratio of AUC^{oral} to AUC^{IV} is then taken as a measure of percent absorption in the gut. From this, absolute bioavailability over a short time frame may be defined as:

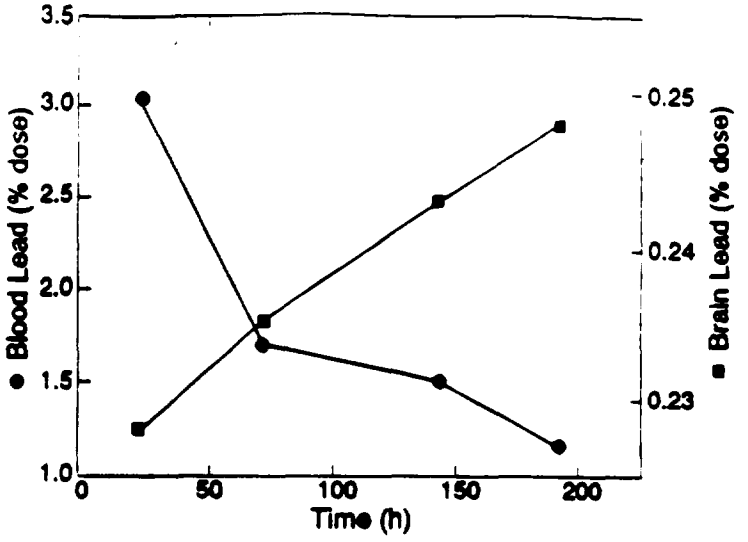


Figure 4-3. **The time-course of bioavailability of lead in the blood (●) and in the brain (■) of juvenile rats following a single dose. Note that accumulation of lead in the target tissue (brain) continues as blood lead decreases. The significance of brain levels indicated is unknown.**

Source: Adapted from Momcilovic and Kostial (1974).

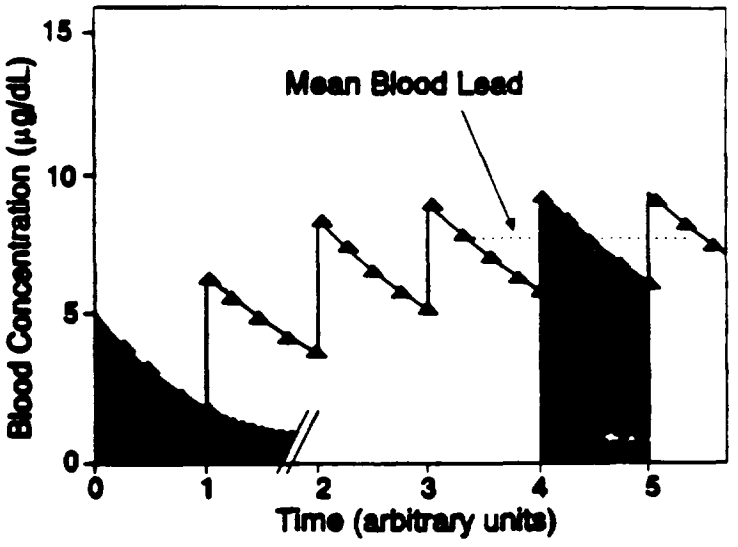


Figure 4-4. **Kinetics of absorption during repeated dosing. At steady state, the area under the curve described by one dosing interval is equivalent to the area under the curve following a single, bolus dose.**

$$\text{Absolute bioavailability} = \frac{(AUC)^{oral}\ (DOSE)^{IV}}{(AUC)^{IV}\ (DOSE)^{oral}} \times 100\%.$$

While careful attention must be given to presystemic elimination (the amount of chemical excreted via the GI tract prior to entry into the systemic circulation), this simple approach can provide, with appropriate sampling and analytical quality control, an effective estimate of the percent absorption into the blood following oral exposure. The longer term kinetics (concentration versus time) of chronic lead absorption are likely to be influenced by the accumulation of lead in peripheral compartments such as bone. Thus, bioavailability estimates conducted with longer-term exposures are preferable in developing quantitative estimates of lead bioavailability. The reader is referred to Gibaldi (1982) for a more detailed discussion of the kinetics of absorption and distribution of toxicants.

4.1.7.5 Absolute Versus Relative Bioavailability

It is usually the case that bioavailability is quantified in absolute terms: it is presumed to be equal to the absorbed fraction for a specific substance. For example, if $CdCl_2$ were 6% absorbed from some medium and CdS were 3% absorbed at equimolar concentrations. the absolute bioavailability for these compounds would be 6 and 3%, respectively.

There are occasions, however, where bioavailability may be specified not in absolute but in relative terms, relative to the bioavailability of some reference compound. Using the earlier examples, if $CdCl_2$ were the reference compound, then the relative bioavailability of the sulfide would be 50% (3%/6% × 100). This approach has much practical value, because one may not have direct bioavailability data for other than one or two forms when estimating risks.

This approach would therefore have value for comparative exposure risk when adjusting risk calculations at Superfund sites. Here, risks are usually calculated from Reference Doses (RfDs) and cancer slope factors that are nearly all based on administered, rather than absorbed, doses. If site-specific exposures involve different chemical/physical forms, it may be necessary to adjust intake dose to uptake dose values in order to account for differing bioavailability in estimating toxicity levels. In such cases, absolute bioavailability measurements may be useful for site-specific forms but are not required for relative risk determinations. While the lead model uses absolute bioavailability as the input parameter, knowledge of the relative bioavailability of ingested materials may be applied. If the relative bioavailability of the material of interest is known relative to a second material whose

absolute bioavailability can be assessed, then the absolute bioavailability of the first can also be estimated.

In addition to establishing the distinction between absolute and relative bioavailability, it is necessary to distinguish between biovailability and solubility. Solubility is a metabolically passive, simplified, in vitro characteristic of a substance that constitutes but one element in bioavailability. This distinction is explored in the following section.

4.1.7.6 Quantitative Experimental Models of Human Lead Bioavailability

Site-specific bioavailability studies of lead in soil have been conducted for several hazardous waste sites in the western United States (LaVelle et al., 1991; Freeman et al., 1991; Weis et al., 1994). In cases where (1) current exposure is significant, (2) soil characteristics preclude simple extrapolation from existing studies, and (3) estimated cleanup costs are sufficiently high, such studies may improve the accuracy and the reliability of the risk assessment process. Site-specific bioavailability studies can be expensive, can require time for completion, and do require considerable technical expertise for the design and conduct of the studies. This means that the remedial project manager (RPM) or risk assessment manager needs to obtain advice from individuals with training and experience in this area. If experimental studies are needed, the toxicology expert may recommend studies at one of the following levels, in order of increasing cost and complexity.

Class I Study

Studies in this class consist of simplified, in vitro approaches in which one determines aqueous solubility of lead from various solid species. This approach has little utility for quantitative human bioavailability assessments. First, solubility itself is but one factor, and a rather crude one, in net uptake of lead from the gut of humans or experimental animals. There are many physiological and biochemical processes occurring in the stomach and the intestines that are not addressed in crude or "beach top" solubility studies. A number of the biochemical factors not reflected in these in vitro, simple solubility approaches were noted by Mushak (1991) and include metal complexing with biochemicals, sustained acid output by the stomach with eating (any material), and uptake processes that are more complex than simple solubilization (e.g., pinocytosis of lead complexed in high molecular weight colloidal particles [micelles]).

A particularly flawed aspect of such in vitro studies is their inability to simulate the kinetically dynamic process that occurs in the intestinal regions (i.e., active transport from intestinal regions via carrier systems [see Section 4.1.5]). Such uptake, thermodynamically

speaking, induces a shift in intraintestinal equilibria among lead forms in the direction of greater dissolution (to compensate for the lead removed by active transport). Such active uptake produces a complex process that yields more bioavailability than predicted in simple in vitro approaches. This shift in equilibrium is compelled by a simple, widely-known principle of chemical processes, Le Chatelier's Principle, that states (CRC, 1978):

> If some stress is brought to bear upon a system in equilibrium, a change occurs, such that the equilibrium is displaced in a direction that tends to undo the effect of the stress.

In the present case, the stress is active intestinal uptake and the displacement to undo the effect is to dissolve more lead during its passage through the gut. Such a shift, relative to a simple bench-top system, is depicted in Figures 4-5 and 4-6.

Class II Study

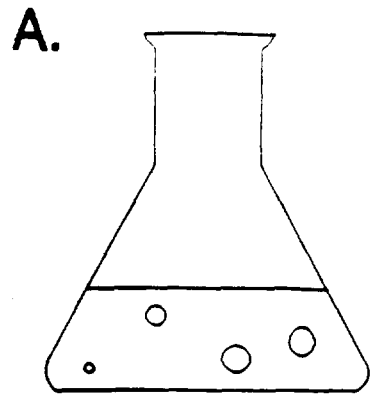
Class II and Class III studies involve in vivo animal models of human bioavailability of lead. They differ in their experimental specifics. Class II investigations are intermediate in vivo studies (i.e., carried out over a relatively short time). Such studies examine the bioavailability of lead within a time frame in which the dosing ends before pseudoequilibrium in the central (blood) compartment is reached. Since lead accumulates in critically important peripheral compartments such as bone and this accumulation will influence longer term uptake and distribution values, longer term studies are desirable for assessing target tissue bioavailability of lead in mammals.

Class II studies are useful in terms of providing a relative index of lead bioavailability, that is, comparison of several lead forms. Class II studies should, of course, consider all the factors already noted that influence any in vivo lead study, including the target population and pathway specifics for the site, age, concentration dependence of lead uptake in the dosing regimen, nutrition, physiology and anatomic structural characteristics.

In terms of model biology, physiology, and behavior, an appropriate selection for human simulation would take account of eating/feeding habits, human versus animal gastrointestinal tract differences, comparative biochemistry, etc.

Class III Study

Bioavailability investigations that have as their purpose the site-specific adjustment of the default bioavailability parameters in the IEUBK model may require a more complex approach. Such advanced studies should only be conducted after consultation with qualified,



$$PbX_{(s)} \rightleftharpoons PbX_{(aq)} \rightleftharpoons Pb^{2+} + X^{2-}$$

Figure 4-5. **Under conditions of equilibrium, the amount of lead as the free ion (Pb^{2+}) is limited by mass balance dissolution of the solid phase (PbX).**

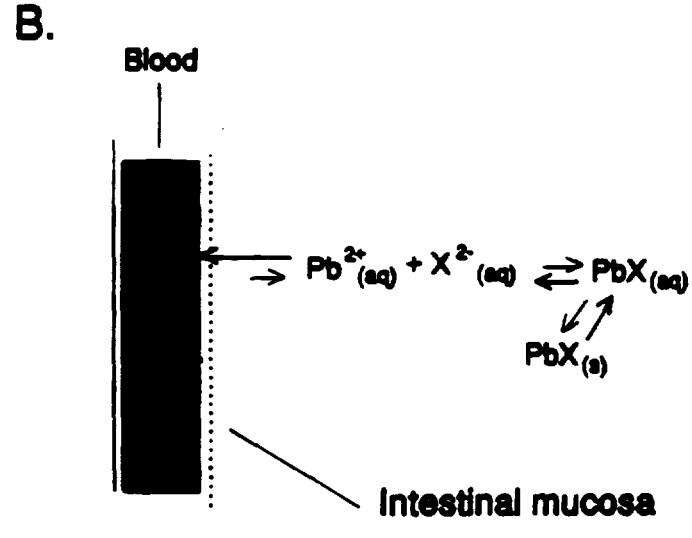


Figure 4-6. **Under physiological conditions, free lead ion (Pb^{2+}) is removed from solution by active and passive absorption mechanisms potentially shifting the equilibrium of the dissolution process far to the left.**

experienced individuals and should be subject to the most rigid quality assurance/quality control (QA/QC) protocols for study management. This especially applies to preserving the original physicochemical form of the lead-containing test materials from a particular site. The design and duration of Class III studies should be such that they assure achievement of near steady state (pseudoequilibrium) for the blood concentration versus time curves. As with Class II studies, Class III investigations need to take account of the site-specific target population and exposure pathways, age of subjects, nutritional and physiological state of the animal, etc.

4.1.7.7 Summary and Advisory Overview for Lead in Soils and Dust

Bioavailability studies are intended to provide valid information about the associations of site-specific physical and chemical properties of exposure media with bioavailability at a target tissue site. Properly designed studies can elucidate differences traceable to such factors as the physicochemical properties of the site's lead-containing media, lead chemical form, matrix species, particle size, mixture effects from other metals or other chemical species from matrix, diet, and such, and study animal or human population variables such as age and levels of exposure. These studies need to meet two fundamental qualifications:

(1) Doses used need to be low enough to be comparable to human exposure situations that are to be assessed. Basing calculations on high doses of lead may greatly weaken the utility of an experimental study.

(2) Animal models need to be carefully examined for their appropriateness to represent human gut processing and absorption of lead. The demonstration that absolute bioavailability is low in an animal model is of limited significance unless that model can be supported as being quantitatively relevant to humans.

Bioavailability factors can be validly adjusted to account for site-specific lead exposure characteristics in the IEUBK model. However, selection of a site-specific bioavailability parameter other than the model default value of 30% for soils and dusts requires considerable caution and warrants review by qualified technical experts.

4.1.8 Bioavailability of Lead in the Diet

The absorption of lead from food and liquid diet by infants up to six months old is known to be very high (Ryu et al., 1983; Marcus, 1989a), and much lower in adults

(Chamberlain et al., 1978; Blake and Mann, 1983; Rabinowitz et al., 1980; James et al., 1985). Less is known about changes in lead absorption from diet for older infants, toddlers, and children. A value of 50% was selected as an intermediate level in children and infants (U.S. Environmental Protection Agency, 1990b).

The exact form of the dietary lead absorption coefficient in humans is not known. There is evidence that the absorption of lead in food by infants is quite high, at least 40 to 50%. The range cited by the U.S. Environmental Protection Agency (1989a) is 42 to 53%. While this probably decreases after infancy, we have no direct evidence on how to interpolate this range for children of ages 2 to 6. A smoothing of the absorption data from infant to juvenile baboon in the studies by Harley and Kneip (1985) has been proposed as a basis for extrapolation by the U.S. Environmental Protection Agency (1989a). In view of the uncertainty about this, we have chosen to keep the same default value of 50% for ages 1 to 6. This value will, at worst, slightly overestimate dietary lead uptake in older children.

Lead absorption from diet depends on the lead concentration in the stomach, and on a host of other dietary cofactors such as zinc, iron, vitamins, and phytate. When dietary lead intake during meals is sufficiently high, absorption of lead through the gut lumen decreases, probably due to competition for the limited anionic lead-binding sites on the gut wall.

The absorption of lead has some similarities with the absorption of other metals (Mushak, 1991), especially alkaline earths such as calcium and strontium. Calcium researchers have hypothesized three possible mechanisms of gut absorption. The first is a type of saturable active transport. This may be a secondary process because the enzyme requiring energy input is on the basolateral membrane and not on the membrane of the gut lumen. It would be more accurate to describe this as a facilitated diffusion process. A second saturable facilitated process involving pinocytic mechanisms has also been hypothesized by calcium researchers, but is not well understood. These saturable diffusion processes are the dominant modes of transport at low concentrations. Processes requiring carriers are often called *facilitated* diffusion processes. For convenience, we may call either of these saturable processes *facilitated* diffusion processes. The third process, the dominant mode of transport at high concentrations, is probably a simple diffusion through tight junctions on the lumenal side and is not saturable. Binding and transport of calcium across the gut lumen involves a protein called calbindin. We have described this as a *passive* diffusion process. The last two processes have no specific inhibitors and are difficult to study. The extent to which lead absorption shares these calcium processes, or is quantitatively different, is not known. The study by Aungst and Fung (1981) on transport of

dissolved lead across the gut lumen in vitro in everted rat intestines shows that lead absorption is likely to consist of two distinct processes. The first process depends on a passive diffusion mechanism that is independent of gut concentration. The second process depends on a facilitated diffusion mechanism that is saturable, with a half-saturation concentration of about 120 μg/L (0.59 μmol). The quantitative extrapolation of this value to human children in vivo is uncertain.

The Glasgow duplicate diet study reported results on infant blood lead and dietary lead intake at a single time point, age 3 months. There appeared to be a very large non-dietary background source contributing about 12 μg/dL blood lead to these infants. This is attributed in part to the inhalation of leaded gasoline, which was still widely used in the United Kingdom, and in part to residual exposure pre-natally. The dietary lead intake in these infants is believed to constitute almost all of the ingested lead, since children at this young age are believed to have minimal contact with soil, house dust, or paint. Some small contribution of inhaled lead particles may be transferred to the ingestion route by mucociliary transport.

A non-linear regression model was fitted to the Sherlock and Quinn data in a form that is directly comparable to the Michaelis-Menten formula used to describe in vitro studies (Aungst and Fung 1981ab). The model that was fitted to all data was:

$$\log(\text{Blood lead}) = \log(B + L * \text{PbIntake} + K * \text{PbIntake} / (1 + \text{PbIntake} / M)).$$

The parameters have the following interpretation:

- B = background lead concentration from pre-natal and inhalation exposure;
- L = linear (passive) uptake coefficient between blood lead and dietary lead intake;
- K = non-linear (facilitated) uptake coefficient between blood lead and dietary lead intake;
- M = Michaelis-Menten type (non-linearity) parameter, the daily dietary lead intake rate at which the facilitated component of lead uptake is half saturated.

Three methods were used to estimate the parameters. The first two methods are based on weights for the grouped data shown in Figure 4-2 of Sherlock and Quinn (1986), shown in this document as Figure 4-1. The first set of weights was based on the estimated sample size within each bar on the graph. The second method was based on the normalized coefficients of variation from the standard error bars for each group. The third method was based on

using within-cell geometric mean blood lead and dietary lead intake values from Table 4-2 in their paper, with cell counts used as weights. The first method appears to be the most accurate, both absolutely and relatively. The fitted blood lead model is:

$$\text{Blood lead} = 10.85 + 0.0090\ \text{PbIntake} + 0.2981\ \text{PbIntake} / (1 + \text{PbIntake} / 90.33)$$

The total blood lead to lead intake regression coefficient at low intake levels (much less than the Michaelis-Menten coefficient M = 90 μg/day) is K + L = 0.0090 + 0.2981 = 0.307 μg/dl per μg/day. The goodness of fit of this non-linear lead uptake model of Michaelis-Menten form to 3-month old human children, combined with the similar piecewise linear model that could be fitted to the water lead studies, and the goodness of fit of the Michaelis-Menten model found for the data on blood lead and lead intake data in infant and juvenile baboons presented by Mallon (1983) support the use of this model for lead absorption in older children as well. The suggestion by Chamberlain (1984) that absorption in adults is greatly reduced at intake rates above 300 μg/d is also consistent with the infant estimate of 90 μg Pb/day.

4.1.9 Bioavailability of Lead in Water

The bioavailability of dissolved lead salts in drinking water is very high when consumed by adults between meals (James et al., 1985), and very low when consumed with meals. The maximum retention of lead in children probably exceeds that of adults, which is about 60% on an empty stomach, and absorption is likely to be only somewhat smaller than retention. Thus the value of 50% is recommended as plausible. A range of values for water lead absorption from the U.S. EPA/OAQPS Staff Paper (1989a), shown in Table 4-1, should be used as a basis for age-variable absorption coefficients.

The volume of water in a typical United States faucet is about 90 to 125 milliliters, and at least two or three faucet volumes must be drawn before the tap water lead concentration decreases to the level of the source water and water distribution line lead concentrations (Schock and Neff, 1988; Gardels and Sorg, 1989; Marcus, 1991a). The sample volume of first-draw water specified in U.S. EPA's drinking water regulation is 1 L (U.S. Environmental Protection Agency, 1991c). Water lead concentrations in most U.S. water supply systems are low (<5 μg/L), but geometric means may exceed 10 to 20 μg/L in first-draw samples from systems with highly corrosive water and a great deal of lead plumbing, which is not uncommon in older urban areas in the northeastern United States.

TABLE 4-1. PIECEWISE LINEAR REGRESSION MODELS FOR BLOOD LEAD VERSUS WATER LEAD IN THREE STUDIES

Parameter	Glasgow Infants N = 91	Edinburgh School Children N = 495	Hawaii Children and Adults N = 180
Intercept μg/dL	12.82	6.84[1]	[1,2]
CONC. for SLOPE CHANGE μg/L	16.4	15.0	15.0
SLOPE (< CHANGE) μg/dL per μg/L	0.254	0.161	0.130
SLOPE (> CHANGE) μg/dL per μg/L	0.0426	0.0318	0.0242

[1]Intercept depends on other covariates.
[2]Not given.

Source: Marcus (1989b) and Maas et al. (1991).

Even if the community mean is low, lead in drinking water in some households may be sufficiently high to cause overt lead poisoning (Cosgrove et al., 1989).

4.1.10 Bioavailability of Lead in Air

Lead on aerosol particles must be inhaled and deposited before pulmonary absorption can occur. Particles inhaled but not deposited may be exhaled or trapped by the mucociliary lift mechanism and ingested. The number of inhaled particles of a given size range varies with the ambient concentration and size distribution and the breathing rate. The breathing rate varies with age and physical activity. Inorganic lead in ambient air consists primarily of particulate aerosols with a size distribution determined largely by the nature of the source and proximity to it. In rural and urban environments, This size distribution is usually from 0.05 to 1 micron. Near point sources, particles greater than 10 microns prevail.

Deposition in the respiratory tract can be by inertial impaction in the nasopharyngeal regions, where the airstream velocity is high, or by sedimentation and interception in the

tracheobronchial and alveolar regions, where the airstream velocities are lower. In the alveolar region, diffusion and electrostatic precipitation also become important.

Particles greater than 2.5 microns are deposited in the ciliated regions of the nasopharyngeal and tracheobronchial airways, where they are passed to the gastrointestinal tract by the mucociliary lift mechanism. Particles small enough to penetrate the alveolar region can be dissolved and absorbed into systemic circulation or ingested by macrophagic cells. Evidence that lead does not accumulated in the lungs suggests that lead entering the alveolar region is completely absorbed (Barry, 1975; Gross et al., 1975). Rabinowitz et al. (1977) found about 90% of the deposited lead was absorbed daily. In the IEUBK model the default assumption is that 35% of the inhaled lead is bioaccessible (reaches the absorbing surface), and 100% of this is absorbed.

4.2 USING THE INTEGRATED EXPOSURE UPTAKE BIOKINETIC MODEL FOR RISK ESTIMATION

4.2.1 Why Is Variability Important?

4.2.1.1 Intent of the Model and the Measure

The Geometric Standard Deviation (GSD) as used in this manual is a measure of the relative variability in blood lead of a child of a specified age, or children from a hypothetical population, whose lead exposures in a specified dwelling are known. *The GSD is intended to reflect the five types of individual blood lead variability identified below, not variability in blood lead concentrations where different individuals are exposed to substantially different media concentrations of lead.*

The IEUBK Lead Model is intended to be used for individual children who live at a residence, or for a hypothetical population of children who may live there in the future, or for hypothetical children who may some day live in a house built on a plot of now vacant land of appropriate size for future construction of a single residential dwelling unit.

4.2.1.2 Individual Geometric Standard Deviation

Why do different children have different blood lead levels? The answer to this question has two parts. The first part of the answer is that children are exposed to different levels of lead in their community environment. The second part of the answer is that individual

children, exposed to exactly the same measured levels of lead, will still have different blood lead levels for the following reasons:

- Different Environmental Context. Carpeting, other furnishings, and accessibility of yard soil affect contact with environmental lead in ways that are not easily measured.

- Behavioral Differences. Interaction with caretakers, with siblings and playmates, and other factors that affect mouthing behavior and play activity will modify lead intake from dust and soil.

- Different Exposures. The children will have different exposures due to differences in contact with soil, dust, water, and other environmental media that vary at different locations and different times, so that no single sample of environmental lead in any medium can be said to completely characterize the child's actual activity-weighted exposure to lead in that medium.

- Measurement Variability. The environmental lead measurements are not perfectly reproducible due to sampling location variability, repeat sampling variability, and analytical method error, so that equality of measured sample lead concentrations does not imply equality of the true exposure concentrations.

- Biological Diversity. Children are biologically diverse so that even children of the same age, weight, and height are expected to have differences in the biokinetic distribution and elimination of lead.

- Food Consumption Differences. A number of factors, including nutritional status and time of ingestion of lead relative to meal times, affect the uptake or absorption of lead ingested from a medium.

While sociodemographic factors underly many of these differences, it is not appropriate to assume any specific effect for future residents. Risk estimates should be applicable to any hypothetical resident, and this requirement adds to the variability associated with the estimate.

4.2.2 Variability Between Individuals Is Characterized by the Geometric Standard Deviation

Inter-individual variability is the starting point for risk analysis using the IEUBK model. Even if we knew the correct value for all of the environmental exposure variables, we could at best predict only the typical blood lead level expected for a child of a certain age who had that exposure. We will therefore assume that individual child blood lead levels can be divided into two parts, a *predicted* blood lead and a *random* deviation from the predicted blood lead level. A statistical model that has proven to be very useful and fits all of the blood lead studies we have analyzed is based on the following three assumptions:

(Assumption 1) Observed blood lead = (Predicted blood lead) * (Random deviation);

(Assumption 2) The random deviation is log-normally distributed with geometric mean or median = 1, and a geometric standard deviation (GSD) defined by GSD = exp (standard deviation of ln (blood lead). Here, exp(.) denotes the exponential function and ln(.) denotes the natural logarithm;

(Assumption 3) The GSD is the same for all values of the predicted blood lead (i.e., for all values of environmental exposure).

Risk is the probability of exceeding the blood lead level of concern. The IEUBK model calculates risk from these three assumptions. The user provides an exposure scenario from which the IEUBK model calculates a predicted blood lead. Then the user provides a blood lead level of concern, whose default value is now defined as 10 μg/dL based on health effects criteria, but can be modified by the user. This risk is calculated as the probability that a standardized, normally distributed random variable exceeds the level Z, where

$$Z = \ln(\text{blood lead level of concern}/\text{predicted blood lead}) / \ln(\text{GSD}).$$

If Z = 1.645, the risk is 5%. If Z = 1.96, the risk is 2.5%. If the GSD is increased, then Z is decreased, and the risk of a blood lead level exceeding the level of concern is increased (provided that the blood lead level of concern is larger than the predicted blood lead, which is usually true). This is illustrated in Figure 4-7. The default value of Z is

$$Z = \ln(10/\text{predicted blood lead}) / \ln(1.6)$$
$$= (2.3026 - \ln(\text{predicted blood lead})) / 0.47.$$

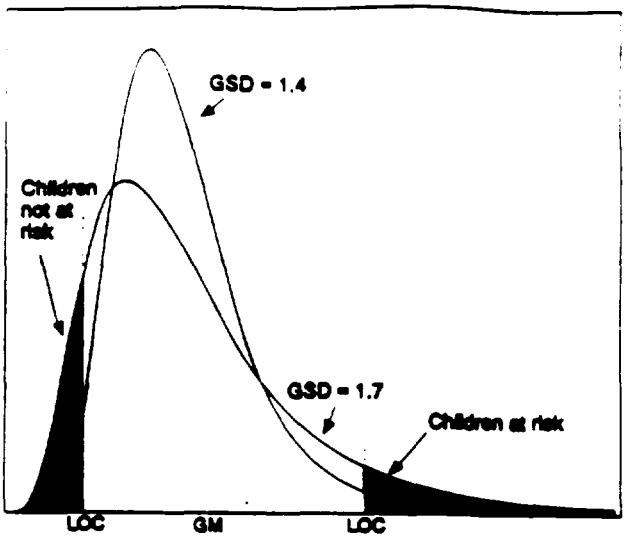


Figure 4-7. The impact of the relative positions of the level of concern (LOC) and the geometric mean (GM) on the proportion of children "at risk" for two populations with different GSDs. If LOC > GM, then the area for children at risk (shaded plus solid) for GSD = 1.7 is greater than the area (solid) for GSD = 1.4. If LOC < GSD, then the area for children *not* at risk (shaded plus solid in lower tail) for GSD = 1.7 is larger than the area for children not at risk (solid in lower tail) for GSD = 1.4.

The GSD has been estimated in a number of ways. The statistical model has the same form as the model used as the basis for estimation of slope factors reported in the Air Quality Criteria Document for Lead (U.S. Environmental Protection Agency, 1986). The GSD values were estimated by exp(s), where s denotes the residual standard deviation of the fitted ln (geometric mean blood lead) as a function of the environmental lead concentrations and of demographic cofactors. The residual standard deviation estimate for ln (blood lead) in a system of structural equations for lead was also used to estimate in some more recent studies.

Estimates of GSD for lead mining and smelter sites have increased towards larger GSD values as the geometric mean blood lead levels at those sites have decreased. This probably reflects the fact that at low to moderate levels of exposure, lead levels are likely to be influenced by several media with similar media-specific uptake rates, rather than by a single dominant medium. This condition tends to magnify individual differences in intake behavior or in biokinetics, and increases the GSD. The GSD estimates for several mining and smelter sites ranged from 1.30 to 1.79 (Marcus, 1992). We chose a value smaller than the

maximum that is consistent with the remaining variability after differences in the usual site specific soil lead and dust lead measurements have been removed. The remaining sources of variability include not only biological and behavioral variability in the children, but also repeat sampling variability, sample location variability, and analytical error. For empirical support in selecting a site specific GSD see Appendix A.

The default value is:

$$GSD = 1.6.$$

This default value is based on calculations of GSDs from specific sites. The median GSDs weighted for sample size within cells were estimated as 1.69 for Midvale, 1.53 for the Baltimore data of the Urban Soil Lead Abatement Demonstration Project, and 1.60 for the Butte study. This type of adjusted GSD calculation was chosen because of its treatment of outliers. Other types of adjusted GSDs, such as those derived from structural analyses are described below.

We must discourage the user from changing the GSD value by use of empirical site-specific data from a blood lead study. As discussed in Section 4.5 below, blood lead studies may be subject to subtle sampling biases and changes in child behavior in response to the study. The GSD value reflects child behavior and biokinetic variability. Unless there are great differences in child behavior and lead biokinetics among different sites, the GSD values should be similar at all sites, and site-specific GSD values should not be needed.

The user may wish to demonstrate that the variability in a specific well-conducted blood-lead study is consistent with the default assumption. In the next section, we will describe how to estimate a site-specific, inter-individual GSD when necessary. These analyses should be done only when necessary, and with thorough documentation of the reasons why the site may have more or less variation among child behavioral and biological parameters than at most other sites. We must remind the user that *it is not necessary to have site-specific blood lead data in order to appropriately use the model with the default GSD*.

4.2.3 Statistical Methods for Estimating the Geometric Standard Deviation from Blood Lead Studies

We have used several statistical methods to estimate GSD values recommended here. Two methods are described in detail in Appendix A. The first method is a direct method in

which environmental lead levels are fixed in ranges or intervals, and blood lead variability for children exposed to these concentrations is calculated directly. The second method is a statistical regression method appropriate to the generally skewed distribution of blood lead values and estimates the variability in blood lead concentrations after an empirical estimate of blood lead concentrations expected at each environmental lead concentration. The two methods give reasonably consistent results. The regression method uses child-specific age and lead concentration. The regression method crudely mimics the IEUBK model.

4.2.4 Choosing the Geometric Standard Deviation: Intra-Neighborhood Variability

There have been some cases in which the IEUBK Lead Model or a preceeding model was used to estimate the distribution of blood lead in a community when only community-level input was available, such as geometric mean soil, dust, and air lead. Further experience with the IEUBK model suggests that this application may be appropriate under some conditions in which certain mathematical assumptions are approximately correct. It also suggests that there are some other situations in which this approach is incorrect because the necessary mathematical assumptions are not satisfied. At this time, we recommend using the IEUBK Lead Model for neighborhood and individual blood lead assessment, but not for communities or for larger scale blood lead assessments without carefully evaluating the input assumptions. The neighborhood scale assessment requires stratifying the neighborhood by intervals of soil and dust lead.

A neighborhood is a spatially contiguous area that often has identifiable physical or geographical boundaries. For the purposes of this manual, a neighborhood is characterized according to the following guidelines:

- Boundaries such as a highway, railroad right-of-way, river, or by non-residential land uses such as commercial, industrial, agricultural, or park;
- Approximately 400 households with about 100 children;
- Church, school, and retail establishments within walking distances;
- Diameter about 1.5 kilometers (1 mile).

The neighborhood concept is used here to classify small areas of relatively similar childhood lead exposure, and will rarely be the same as a census tract, political locale such as a precinct or ward, or community association membership area.

Input parameters for the model at a neighborhood scale should be some measure that characterizes typical exposure concentration in a medium, such as the arithmetic mean or geometric mean, or the median. When activity pattern or behavior weighted exposure information is unavailable, we recommend use of the arithmetic mean to characterize soil lead concentrations in areas that are sufficiently small that any part of the area may be accessible to a typical child living at a random residence located within the area. This will certainly be applicable to the yard and adjacent play areas of a single residence.

Our recommended approach for risk estimation involves more calculations than the single-input soil and dust lead, but much less calculation than the use of each individual yard or housing unit. Our approach requires the division of the neighborhood into units that are larger than single yards or other sites, but smaller than the whole neighborhood, and clearly must depend on the scale of a risk assessment. Risk within a neighborhood can be assessed in a single model run only if media concentrations of lead are relatively homogeneous between different residential sites.

There is no definition of a "community" for model use. It is expected that older children will be able to play anywhere within a neighborhood, but are limited to their own neighborhood within the community. An alternative approach is to define "neighborhoods" by isopleths or contours of soil lead concentrations, but this is more likely to be useful in the vicinity of active or inactive smelter or battery recycling plants, where soil lead deposition has a definite point source pattern. No specific approach based on Geographic Information Systems (GIS) data bases has yet been adopted. The definition of neighborhood scale suggested here is roughly equivalent to an area of 4 to 10 city blocks in many urban areas (160 to 240 meters square). A neighborhood should not be larger than a one kilometer square.

4.2.5 Basis for Neighborhood Scale Risk Estimation

The basis of the neighborhood approach is that a few important environmental parameters largely determine the predicted geometric mean blood lead. Since the environmental lead concentrations are known to have some measurement error, there should be little loss of accuracy in grouping the environmental lead concentrations by small

intervals. For example, the interval ranges for soil lead concentration could be 0 to 249 $\mu g/g$, 250 to 499 $\mu g/g$, 500 to 749 $\mu g/g$, etc. Soil lead levels in an interval, for example from 250 to 499 $\mu g/g$, would be described by a single number in that range, such as the midpoint of the interval at 375 $\mu g/g$.

One of the most important determinants of blood lead concentration in children is lead in household dust. It is necessary to use small intervals of dust lead concentration along with small intervals of soil lead concentration. There are many other sources of lead in household dust in addition to soil lead, including dust lead from air lead deposition, from interior lead-based paint, and from workplace dust carried home by adults residing in the house. The actual range of dust lead concentrations corresponding to a soil lead interval is therefore generally much wider than the range of soil lead concentrations.

There may be circumstances in which other lead exposures in a neighborhood are known, and vary over a wide range. For example, there may be information on water lead concentrations in different houses. Some of the houses may have sufficiently high water lead concentrations that lead in water becomes another significant source of lead exposure. Additional stratification or classification of sites by this variable may also be useful.

Neighborhoods defined by small geographic areas are also much more likely to be homogeneous with respect to sociodemographic factors that affect blood lead variability. There should be some similarity in child activity patterns, household environmental contexts, behavioral patterns, and nutritional patterns within a neighborhood. Therefore, the individual GSD may be applied plausibly to the relatively homogeneous subpopulation within a neighborhood. If the neighborhood defined initially is very heterogeneous, then a larger GSD may be needed. It would be better to subdivide the neighborhood defined initially into more homogeneous subareas. This requires knowledge about the neighborhood residents, or an assumption about future residents.

4.2.6 Relationship Between Geometric Standard Deviation and Risk Estimation

The GSD is a very sensitive parameter for risk estimation. In this model, we use "risk" in the following specific ways:

- Individual risk is the probability that a hypothetical child living in a particular house or dwelling unit characterized by its environmental lead levels will have a blood lead concentration that exceeds a user-specified level of concern;

- Neighborhood or community risk is the fraction of children in a neighborhood or community characterized by a specified distribution of environmental lead concentrations that are expected to have blood lead concentrations exceeding a user-specified level of concern.

The assessment of potential health risk from environmental exposure to toxicants is one of EPA's most significant activities. We are using only part of this process. An elevated blood lead concentration (however one defines "elevated") is an index of internal exposure or body burden of lead. It is a useful index precisely because it changes in response to changes in exposure, with characteristic time scales of a few days or so in plasma and red blood cells, reflecting deeper changes of a few months in soft tissues, and years in hard bone. An elevated blood lead concentration is not precisely an adverse health effect by itself, but has been a very useful predictor of an increased likelihood of neurobehavioral deficits in children. The "risk" involved here is the risk of an increase in an easily measured index of lead exposure that is a predictor of adverse health effects.

The most general form of the model is multiplicative:

Blood lead = controllable factors * random factors.

For a single child, with defined sources of exposure, the IEUBK model estimates the geometric mean blood lead, or typical blood lead (i.e., the median when variability is log-normal, as it usually is). The model then is given by:

Blood lead = GM * exp(Z * ln(GSD))

where GM is the model-predicted geometric mean blood lead, exp(.) is the exponential function, ln(.) is the natural logarithm function, and Z is a normally distributed random variable. Therefore risk, defined as a probability for a single child, is calculated by the equation

Risk = Probability{Blood lead > level of concern for given exposure}

$$= \text{Probability}\{Z > (\ln(\text{level of concern}) - \ln(GM)) / \ln(GSD)\}.$$

When the level of concern is greater than the expected or typical blood lead at that exposure, then risk increases when GSD increases. Figure 4-7 illustrates the difference in "at risk" children for two populations, one with a GSD of 1.4 and another with 1.7. When the level of concern is above the geometric mean, the population with the higher GSD has a greater proportion of the children at risk. When the level of concern is less than geometric mean, the population with the lower GSD has a greater proportion of children at risk.

4.2.7 Risk Estimation at a Neighborhood or Community Scale

4.2.7.1 What Do We Mean by "Neighborhood" or "Community" Risk?

Representative questions of interest in assessing the risk of elevated child blood lead in a neighborhood are:

- What is the frequency distribution of risk of exceeding a blood lead concentration of concern, such as 10 μg/dL, within the neighborhood?

- What fraction of a hypothetical or actual population of children would be expected to exceed some specified blood lead concentration of concern if they resided in the representative sample of houses in this neighborhood for which we have soil and dust lead data?

- How much could we reduce high individual risk or the fraction of children with elevated blood lead concentrations by cleaning up soil to some specified level?

- What is the distribution of risks for a hypothetical population of children if housing units were constructed on soil at this vacant site?

The implicit definition of risk in these questions is the fraction of children living in a dwelling unit anywhere in the neighborhood who have elevated blood lead levels. We see that the neighborhood or community risk level has two distinct components of variability:

(1) Inter-individual differences, as in Section 4.2.4; and
(2) Inter-dwelling unit differences in lead exposure.

In some circumstances, these two can be combined and the same approach used to estimate the fraction of children at risk in a neighborhood. But, if there is a broad distribution of inter-dwelling unit differences, as is commonly observed, then a simplistic application of the IEUBK model may substantially under-estimate the real risk from the most contaminated parts of the neighborhood. Whatever the distribution of inter-dwelling unit or intra-neighborhood exposure levels, the "sum of risks" approach can always be applied. Note that there is a subtle difference between inter-dwelling exposure and intra-neighborhood exposure. Inter-dwelling exposure distribution would be the distribution of exposures measured in each home and would assume that the individual exposure is within the property boundaries of the dwelling unit. Intra-neighborhood exposure would include additional exposure from nonproperty sources, such as parks, schools and playgrounds.

4.2.7.2 Neighborhood Risk Estimation as the Sum of Individual Risks

Neighborhood risk is based on the expected number of children in the neighborhood who have elevated blood lead levels, here taken as greater than 10 μg/dL. Using the computer model, some of these questions can be addressed by the following procedure:

1. Set up a batch mode file in which each line represents the age and environmental lead exposure of each child in the real or hypothetical population.

2. Use the IEUBK Lead Model to estimate the geometric mean blood lead for each child in the batch mode file.

3. Apply an individual GSD to estimate the probability of exceeding the blood lead level of concern for each child or each household in the batch mode file.

4. Calculate the expected number of blood lead values exceeding the level of concern by adding up the probability of exceeding the blood lead level of concern across all children in the batch mode file.

   Note that even houses with low lead concentrations have a small positive risk for resident children. In houses with high lead concentrations, the risk of elevated blood lead is much larger, but some children (even in those high lead houses) will not have elevated blood lead concentrations. The total of all such risks characterizes neighborhood exposure.

5. Neighborhood risk is the ratio of the calculated expected number of blood lead values exceeding the level of concern to the total number of children in the batch mode file. This last point is illustrated in the following narrative.

4.2.7.3 An Example for the "Sum of Individual Risks" Approach

Suppose that there are data on four households with children in a neighborhood. Residents of each household are exposed to lead-contaminated soil. The first house has 250 μg/g lead in soil, the second has 250 μg/g, the third has 1000 μg/g, and the fourth house has 1000 μg/g. We have assumed dust lead concentrations as 70 percent of the soil lead concentration in houses 2 and 4, and as 15 percent of the soil lead concentration in houses 1 and 3. We have added 10 μg/g to dust lead as an estimate of the air lead contribution to dust lead at 0.1 μg Pb per cubic meter of air. The respective dust lead concentrations are thus 47.5 μg/g, 185 μg/g, 160 μg/g, and 710 μg/g.

The neighborhood is usually not just 4 houses. We may have samples at only these 4 houses, or there may be 100 houses at each of these 4 soil and dust lead concentrations. The assumption is that the samples are representative of the exposure distribution in the neighborhood. We are showing calculations for four houses only for the purposes of illustration. The risk estimates are intended to be unbiased estimates of potential risk for other years in which different children, not in the current sample, may occupy the same or other houses in the neighborhood. Obviously, a reliable estimate of neighborhood risk will require many more than 4 houses.

All other parameters are set to default values. We used a soil and dust absorption model with 30% absorption of lead from both dust and soil. (Smaller values of soil lead absorption may be needed for some sites—see Section 4.1). We assumed GSD = 1.6; larger values of GSD may be needed at some sites. The probability density of blood lead for four houses is shown in Figure 4-8.

For the house with soil lead at 250 μg/g and dust lead at 47.5 μg/g, we expect 0.55% of children to exceed 10 μg/dL. For the house with 250 μg/g soil lead and 185 μg/g dust lead, we expect 1.99% to exceed 10 μg/dL. For the house with soil lead at 1,000 μg/g and dust lead at 160 μg/g, we expect 21.06% of children to exceed 10 μg/dL. For the house with 1000 μg/g soil lead and 710 μg/g dust lead, we expect 42.68% to exceed 10 μg/dL. The sum of the risks for these four houses is 0.55% + 1.99% + 21.06% +42.68% = 66.28% children = 0.6628 children expected to exceed 10 μg/dL, or an average risk for the neighborhood of 66.28% / 4 = 16.57%, which is greater than the 5% neighborhood risk

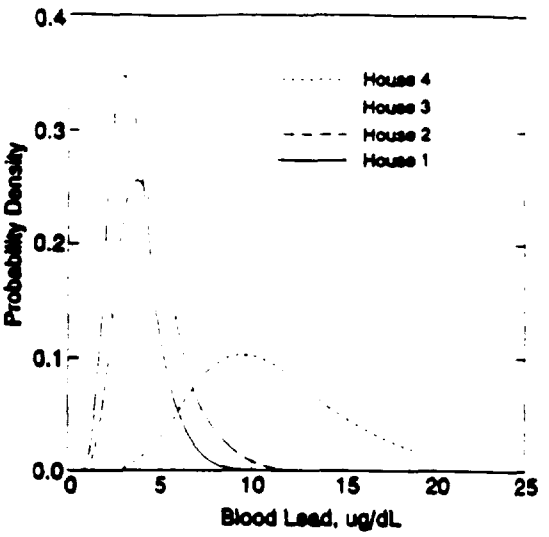


Figure 4-8. Probability density of blood lead in houses 1 to 4.

target used in this example. However, the major part of the risk falls in the one house with high soil and dust lead concentrations.

The use of aggregate neighborhood input data requires that we compare the probability density function (PDF) and elevated blood lead (EBL) risk calculated from aggregate parameters with the correct PDF and EBL risk functions, which are the mathematical composites of the individual PDF and risk functions. Expressed mathematically:

$$\text{true neighborhood PDF} = (\text{PDF(site 1)} + \text{PDF(site 2)} + \ldots)/N$$
$$\text{true neighborhood risk} = (\text{risk(site 1)} + \text{risk(site 2)} + \ldots)/N \qquad \text{(Equation 4-1)}$$

The approach we have outlined here does not require any mathematical assumptions about the distribution of soil and dust lead concentrations, nor of any other parameters or variables except for blood lead. We have assumed that the conditional distribution of individual blood lead is log-normal with a constant GSD (given specified values of lead exposure variables that determine the geometric mean blood lead for individuals with that exact environment). The method suggested here is the most convenient and flexible framework we have found for neighborhood assessment of the effect of soil lead abatement.

4.2.7.4 Assessment of Risk Using Grouped Data for a Neighborhood

The example in the preceding section had a "neighborhood" with only 4 houses, so that the amount of work required was not very burdensome. In the real world, the site manager or risk assessor may be dealing with relatively homogeneous neighborhoods or small communities with several hundred households. These calculations can be simplified by grouping soil and dust lead levels into small cells with fixed ranges of values. The grouped data within each cell are all assigned the same value, such as the midpoint of the interval.

Each cell is then assigned a statistical weight. The statistical weights could be:

(1) The number of housing units with soil and dust lead concentrations in the interval;

(2) The number of children observed or expected to live in housing units with soil and dust lead concentrations in the interval;

(3) The fraction of housing in a neighborhood that is expected to have soil and dust lead concentrations in the interval;

(4) The fraction of area in as-yet-undeveloped neighborhoods with soil and dust lead concentrations in the interval.

The probability density function (PDF) and risk of EBL children in then the weighted sum of the cell PDF or cell risks. If the respective weights are denoted weight (cell 1), weight (cell 2), etc., and the PDFs are denoted PDF (cell 1), PDF (cell 2), etc., and the risks are denoted risk (cell 1), risk (cell 2), etc., then:

$$\text{neighborhood PDF} = [\text{weight (cell 1)} * \text{PDF (cell 1)} + \text{weight (cell 2)} * \text{PDF (cell 2)} + \text{etc.}] / [\text{weight (cell 1)} + \text{weight (cell 2)} + \text{etc.}]$$

$$\text{neighborhood risk} = [\text{weight (cell 1)} * \text{risk (cell 1)} + \text{weight call (cell 2)} * \text{risk (cell 2)} + \ldots] / [\text{weight (cell 1)} + \text{weight (cell 2)} + \ldots]$$

The following hypothetical example may illustrate these points. Suppose that a random sample of 250 houses and apartments has been obtained in a neighborhood. The number of houses in each interval of 250 $\mu g/g$ soil and 250 $\mu g/g$ dust lead is shown in Table 4-2. This

TABLE 4-2. EXAMPLE OF NEIGHBORHOOD RISK ESTIMATION WITH GROUPED DATA

Hypothetical example of grouped data for a neighborhood with dust and soil samples of 250 sites of house yards. Intervals are 250 μg/g in soil and in dust lead.

Soil Interval	Soil Midpoint	Dust Interval	Dust Midpoint	Statistical Weight	Blood Lead[1] (μg/dL)	Risk[2] Percent
0-249	125	0-249	125	30	2.9	0.39
0-249	125	250-499	375	50	4.3	3.45
0-249	125	500-749	625	20	5.7	10.61
250-499	375	0-249	125	10	4.1	2.70
250-499	375	250-499	375	40	5.4	9.36
250-499	375	500-749	625	30	6.7	18.62
250-499	375	750-999	875	20	7.9	28.52
500-749	625	250-499	375	10	6.5	16.45
500-749	625	500-749	625	20	7.7	26.86
500-749	625	750-999	875	10	8.8	38.16
500-749	625	1000-1249	1125	3	9.9	47.56
750-999	875	1000-1249	1125	4	10.8	52.78
750-999	875	1750-1999	1875	1	13.6	72.73
1000-1249	1125	1250-1499	1375	2	12.5	66.93
TOTAL				250		14.28

[1]Calculated from IEUBK model with default parameters.
[2]Assuming GSD = 1.6.

TABLE 4-3. EXAMPLE OF NEIGHBORHOOD RISK ESTIMATION WITH COARSELY GROUPED DATA

Hypothetical example of grouped data for the same neighborhood as in Table 4-1, with intervals of 500 μg/g in soil and dust lead.

Soil Interval	Soil Midpoint	Dust Interval	Dust Midpoint	Statistical Weight	Blood Lead[1] (μg/dL)	Risk[2] Percent
0-499	250	0-499	250	130	4.2	3.05
0-499	250	500-999	750	70	6.8	19.81
500-999	750	0-499	250	[illegible]	6.4	15.45
500-999	750	500-999	750	30	8.7	36.05
500-999	750	1000-1499	1250	7	10.9	55.50
500-999	750	1500-1999	1750	1	12.8	66.92
1000-1499	1250	1000-1499	1250	2	12.4	64.01
TOTAL				250		14.41

[1]Calculated from IEUBK model with default parameters, ages 6 to 84 mo.
[2]Assuming GSD = 1.6.

same example is shown in Table 4-3 in intervals of 500 μg/g in soil and 500 μg/g in dust. There is no requirement that there be equal interval lengths in either soil or dust.

The user may then calculate neighborhood risk in three ways:

- Sum of risks for 250 housing units;
- Sum of risks for 14 cells or groups of width 250 μg/g soil and dust;
- Sum of risks for 7 cells or groups of width 500 μg/g in soil and dust.

The results of calculations are shown in the Tables 4-2 and 4-3. The total risk in Table 4-3 is calculated as:

$$(130 * 3.05\% + 70 * 19.81\% + 10 * 15.45\% + 30 * 36.05\% + 7 * 55.50\% + 1 * 66.92\% + 2 * 64.01\%)/250 = 14.41\%$$

The risk calculation in Table 4-2 is similar. If there are not too many cells, the amount of calculation can be strikingly reduced. However, as the intervals are made longer, there is a corresponding loss of accuracy in the neighborhood risk estimate. The extra effort in calculating risks with 250 μg/g intervals (14 cells) is probably compensated by the increased precision, with an estimate of 14.28% instead of 14.41%. The actual risk for the ungrouped sample with 250 simulated houses in 14.13%.

4.2.7.5 Assessment of Risk with Neighborhood or Neighborhood-Scale Input

There are situations in which it is either inconvenient or impossible to apply the IEUBK model at the intended household residence scale. For example, if only neighborhood mean values or geometric mean values of input parameters such as soil and dust lead are available, the model estimate may be far less reliable than if individual residential measurements were made. Another possibility is that there are a substantial number of soil and dust lead measurements at a site, but not at houses or locations within the site where blood lead and EBL risk estimates are needed, for example, to compare with blood leads observed at residences where there are no environmental data. There are some circumstances in which this is clearly not a valid application of the model. As we do not clearly understand the range of conditions under which the IEUBK model may be used with large-scale input data at this time, we must discourage use of the IEUBK model except with single-residence or residential lot-sized input data, or with data grouped into cells as in Section 4.2.7.4.

4.3 ENVIRONMENTAL PATHWAY ANALYSIS

4.3.1 Concept of Pathway Analysis

Environmental pathways for lead have been a subject of interest for EPA for a long time. Methods for analyzing with multi-media exposure pathways from air lead were used in developing slope factors for blood lead versus air lead, dust lead, and soil lead in EPA's Air Quality Criteria document (U.S. Environmental Protection Agency, 1986). Even though the focus was on exposure to air lead as a primary source, it was clearly recognized that whatever the source of lead in air, paint, or soil, the primary exposure vector for young children was through fine particles of surface soil and household dust that adhered to the children's fingers and were ingested in the course of normal hand-to-mouth contact at ages one to five years. Thus the total impact of air lead exposure had to be evaluated as the sum of exposure over several pathways (Figure 4-9).

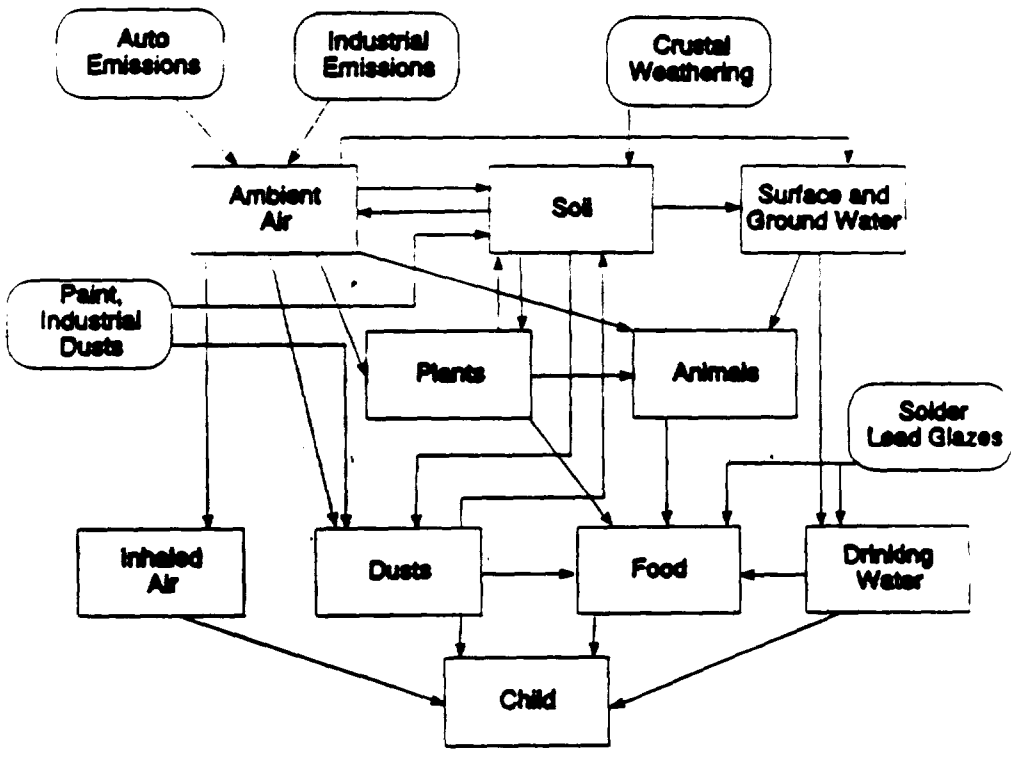


Figure 4-9. Exposure pathways of lead in the environment.

The IEUBK model assists the user in defining critical pathways for each exposure scenario. For example, there are two places on the Multiple Source Analysis Menu for household dust where pathway information may be inserted:

(1) the soil-to-to-dust pathway coefficient may be entered in the first line of the Multiple Source Analysis Data Entry Menu, replacing the default value of 0.70 μg/g dust per μg/g soil lead;

(2) the direct air-to-dust pathway coefficient is on the second line of the Multiple Source Analysis Data Entry Menu, replacing the default value of 100 μg/g dust lead per $\mu g/m^3$ air Pb.

The following paragraphs provide the basis for some of the default parameters used in the IEUBK model, and suggest some methods for estimating alternate coefficients from site-specific data, provided the user has some knowledge of statistical regression. While physical measurement methods such as a comparison of stable lead isotope composition ratios have been used for source apportionment studies (Yaffee et al. 1983; Rabinowitz 1987), most users will probably have to infer site-specific pathway parameters by statistical analyses of field data.

4.3.2 Pathway Analyses by Linear Regression

The slope factor approach, described in Section 1.5, determines the linear relationship between two pathway components. This methods was used for the EPA Exposure Analysis Methodology Validation (U.S. Environmental Protection Agency, 1989a) to show that there is a clear relationship between lead in air (PbA), lead in soil (PbS), and lead in dust (PbD). This relationship may be approximately linear, depending on properties of soil and dust lead particles; if not linear, then it is at least a positive cause and effect relationship. The relationship was established using data from air lead point sources such as primary and secondary lead smelters, other non-ferrous metal smelters, and lead battery plants. The analysis, using mean values, found the relationship:

$$PbD = b_{D0} + b_{DA}\ PbA + b_{DS}\ PbS \qquad \text{(Equation 4.3-2)}$$

where b_{DS} = 0.364 for all point source communities, but = 0.894 for the East Helena primary lead smelter community. This suggests that there may be substantial differences among communities in terms of soil-to-dust transfer.

The direct air-to-soil relationship was also estimated:

$$PbS = b_{S0} + b_{SA}\ PbA \qquad \text{(Equation 4.3-3)}$$

When estimating slope factors by a sequence of regression equations the user should be aware that the "measurement errors" in pathway equations will almost certainly attenuate the size of the regression coefficients, and could even reverse the sign of the coefficients (Kupper 1984). Structural equation modeling techniques attempt to resolve this problem by the simultaneous estimation of coefficients in pathway models in the face of measurement errors.

4.3.3 Pathway Analysis Using Structural Equation Models

Systems of linear equations in which the output of one equation (such as PbD predicted from PbS and XRFI) is used as the input or predictor in another equation (such as PbB from PbD) can be reliably estimated using a method known as structural equation models (Bollen 1990). This method was introduced in the analysis of blood lead data by Bornschein et al. (1985) and Clark et al. (1985) in their analyses of data from the Cincinnati Prospective Childhood Lead Study. Several authors have extensively explored applications of the method to environmental lead data (Marcus 1991; Burgoon and Menten 1991). Two different approaches were compared, and found to produce very similar results.

The first approach uses linear equations without logarithmic transformation, but with a robust method of estimation that is not sensitive to skewness or to instability of measurement error variances. (The software implementation in the EQS program (Bentler 1989) was particularly convenient.) For a lead mining community, or an urban area in which air lead levels are so low as to be negligible predictors of blood lead, A typical small system of equations might be:

$$PbB = b_{B0} + b_{BS}\ PbS + b_{BD}\ PbD + b_{BXI}\ XRFI \qquad \text{(Equation 4.3-6)}$$

$$PbD = b_{D0} + b_{DXI}\ XRFI + b_{DS}\ PbS \qquad \text{(Equation 4.3-7)}$$

$$PbS = b_{S0} + b_{SXE}\ XRFE + b_{Sage}\ House\text{-}age \qquad \text{(Equation 4.3-8)}$$

where: PbB = blood lead concentration (μg/dL)

PbS = soil lead concentration ($\mu g/g$)
PbD = dust lead concentration ($\mu g/g$)
XRFI = interior measurement of lead-based paint by XRF (mg/cm^2)
XRFE = exterior measurement of lead-based paint by XRF (mg/cm^2)
b_{nm} = raw regression coefficient where the subscript refers to: response variable n on predictor m,
B0 = intercept
BD = blood to dust
BXI = blood to XRFI
D0 = intercept
DXI = dust to XRF (interior)
DS = dust to soil
S0 = intercept
BS = blood to soil
SXE = soil to XRF (exterior)
Sage = soil to house age

This model assumes that there is direct ingestion of interior lead paint, which also contributes to household dust, but no direct ingestion of exterior lead-based paint. The exterior lead-based paint contribution is subsumed in the paint to soil to dust pathway. Because of the linear equation formulation, partial effects of lead source terms are preserved:

$$PbB = c_{B0} + c_{BS}\, PbS + c_{BXI}\, XRFI \qquad \text{(Equation 4.3-9)}$$

$$PbB = d_{B0} + d_{BXE}\, XRFE + d_{BXI}\, XRFI \qquad \text{(Equation 4.3-10)}$$

$$c_{BS} = b_{BS} + b_{BD}\, b_{DS} \qquad \text{(Equation 4.3-11)}$$

$$d_{BXI} = b_{BXI} + b_{BD}\, b_{DXI} \qquad \text{(Equation 4.3-12)}$$

$$d_{BXE} = (b_{BS} + b_{BD}\, b_{DS})\, b_{SXE} \qquad \text{(Equation 4.3-13)}$$

where: c_{Bm} = composite regression coefficient for blood on predictor m
d_{nm} = composite regression coefficient for indirect pathways from predictor m to response n

The second approach uses logarithmic transformation of the equations in the system Equations 4.3-6 through 4.3-8:

$$\log(PbB) = \log(b_{B0} + b_{BS}\ PbS + b_{BD}\ PbD + b_{BXI}\ XRFI) \quad \text{(Equation 4.3-14)}$$

$$\log(PbD) = \log(b_{D0} + b_{DXI}\ XRFI + b_{DS}\ PbS) \quad \text{(Equation 4.3-15)}$$

$$\log(PbS) = \log(b_{S0} + b_{SXE}\ XRFE + b_{Sage}\ House\text{-}age) \quad \text{(Equation 4.3-16)}$$

This system can be estimated using SAS PROC MODEL or similar programs for non-linear systems modelling. All of the coefficients were constrained to be non-negative, since negative coefficients are non-interpretable. However, the appearance of a negative estimate for an intrinsically positive coefficient should be taken as a diagnosis of some statistical problem, such as multi-colinearity or the omission of important predictive variables.

4.3.4 Regression Analyses for Multiple Exposure Pathways: Soil Example

The variables for regression analyses were described briefly in Section 1.5. The use of a regression coefficient in risk assessment is a complicated matter, because one can use either *aggregate* regression coefficients, which combine information on all exposure pathways, or *disaggregate* regression coefficients in which each exposure pathway has its own slope coefficient. The exposure of young children to air lead includes soil and dust pathways, as well as direct inhalation. This is discussed in detail in the OAQPS staff papers (U.S. Environmental Protection Agency, 1989ab) based on earlier work by Brunekreef (1984). The aggregate blood lead regression coefficient for air lead, including soil and dust exposure pathways, is c_{BA} = 5 to 6 μg Pb/dL blood per μg Pb/m^3 air, whereas the direct inhalation coefficient b_{BA} is only about 2 μg Pb/dL blood per μg Pb/m^3 air. For a simple soil lead pathway model,

soil → dust → hands → child
soil → hands → child

whose equations are given by

$$PbB = b_{B0} + b_{BS}\ PbS + b_{BD}\ PbD \quad \text{(Equation 4.3-17)}$$

$$PbD = b_{D0} + b_{DS}\ PbS \quad \text{(Equation 4.3-18)}$$

the aggregate blood lead vs. soil lead regression coefficient should be

$$c_{BS} = b_{BS} + b_{BD}\, b_{DS} \qquad \text{(Equation 4.3-19)}$$

An empirical regression coefficient approach would use only the three coefficients b_{BS}, b_{BD} and b_{DS}. In the absence of data from a well-conducted child blood lead study at the same site or at some similar site, including both soil and dust lead data matched to each child's total lead exposure, there is no basis for calculating the aggregate soil lead coefficient. However, the use of a model like the IEUBK model allows estimation of the parameters b_{BS} and b_{BD} in Equation 4.3-8 from a synthesis of many diverse studies and does not require blood lead data at the site. Any additional information about site-specific exposure and soil or dust lead characteristics would progressively refine the model predictions, even without a child blood lead study. Site-specific soil and dust lead data are needed in either approach. The IEUBK model has a parameter in the Multiple Source Analysis for Dust in which the soil-to-dust coefficient b_{DS} can be inserted.

4.4 USE OF DATA FROM BLOOD LEAD STUDIES

4.4.1 Overview

In general, data from well-conducted blood lead studies of children at a site can provide useful information to the risk assessor and site decision maker. The purposes of this chapter are to explain what type of information a well-conducted blood lead study can provide, how blood lead study data can be used when assessing exposure to lead, and how to interpret model predictions when blood lead data for a site are also available.

Proper design and conduct of a blood lead study are critical if the results of the study are to be considered by the risk assessor. Blood lead data alone, without environmental lead exposure data and without elements of study design that control rapid changes in exposure prior to sampling, or without adequate control for sampling and analysis, should not be used to assess risk from lead exposure or to develop soil lead cleanup levels. However, a well-designed and conducted blood lead study can be useful in conjunction with site-specific environmental data in evaluating risk to children.

Blood lead concentrations are widely held to be the most convenient, if imperfect, index of both lead exposure and relative risk for various adverse health effects (U.S.

Environmental Protection Agency, 1986). In terms of exposure, however, it is generally accepted that blood lead concentrations yield an index of relatively recent exposure because of the rather rapid clearance of absorbed lead from the blood. Such a measure, then is of limited usefulness in cases where exposure is variable or intermittent over time, as is often the case with pediatric lead exposure.

According to the EPA Science Advisory Board in its 1992 report, "Review of the Uptake Biokinetic Model for Lead" (U.S. Environmental Protection Agency, 1992a), blood lead concentrations are responsive to abrupt or unanticipated changes in recent lead exposure for children. Since internal exposure is a function of lead intake (concentration multiplied by intake rates) and uptake, these changes can be environmental, behavioral, and physiological. For example, leaving a child in a house where lead-based paint has just been sanded would likely result in a significant elevation in that child's blood lead concentration. Reduction in a child's blood lead concentration may result from altered behavior that reduces exposure to lead (i.e., more frequent house cleaning, more attention to child's cleanliness, etc.). Cross-sectional blood lead studies (all done within a short time interval) are most useful when there is no reason to believe that child lead exposure has changed significantly within the last few months due to changes in environment or behavior.

A blood lead value may say little about any excessive lead intake at an early age, even though early childhood exposure may have resulted in significant irreversible toxicity. On the other hand, analyses that are retrospective in nature such as whole tooth or dentine analyses can only be done after the particularly vulnerable age in children—under 4-5 years—has passed. Such a measure, then, provides little basis upon which to implement regulatory policy for environmental or clinical intervention.

Furthermore, over a relatively broad range of lead exposure through some medium, the relationship of lead in the external medium to lead in blood is curvilinear, such that relative change in blood lead per unit change in exposure level generally becomes increasingly less as exposure increases. This behavior may reflect changes in tissue lead kinetics, reduced lead absorption, or increased excretion. In any event, modest changes in blood lead concentrations with exposure at the higher end of this range are in no way to be taken as reflecting correspondingly modest changes in body or tissue uptake of lead, (U.S. Environmental Protection Agency, 1986).

Data from good quality blood lead studies can be useful in examining the predictiveness of the model. The IEUBK Model predicts blood lead concentrations in children younger

than 84 months based on environmental inputs for soil, house dust, water, air, and dietary lead intake. It would be logical to assume that the distribution of blood lead concentrations predicted by the model using site-specific data would be generally similar to those measured in the population, provided that the actual blood lead study was well designed and conducted. The IEUBK Model may not be able to account for all sources of exposure. If the predicted blood lead concentrations are not similar to those observed, an attempt should be made to identify the reasons for those differences.

It is important to recognize that most implementations of the lead model now rely on the assumption that exposure to lead in soil and dust is primarily residential in nature. However, in an actual population of children, there will be substantial opportunity for non-residential lead exposures. Periods of time spent away from the home may also have the effect of reducing the residential exposures that would otherwise occur. The fact that the model applications cannot now track all aspects of nonresidential of lead sources that a child may encounter implies that a precise match between calculated and predicted blood lead distributions cannot be expected. Nevertheless, due to the importance of residential exposures to lead in children, a reasonable overall agreement should be anticipated in such comparisons. These considerations argue that reliance on P-values from statistical tests is not an appropriate basis for judging the comparability between observed and predicted blood lead concentrations. It should be noted that calculations of blood lead concentrations on the assumption of residential exposure is a useful endpoint in site risk evaluation, as many children will indeed experience primarily residential exposures to lead.

It is important to understand that the model should not necessarily be expected to reproduce the observed blood lead concentrations exactly. The model predicts the geometric mean blood lead level corresponding to a given set of exposure inputs. Probability distribution estimates produced by the model for a given GSD can be used to define a prediction interval for blood lead concentrations. As long as the interval includes the observed blood lead corresponding to the same exposure inputs, the model has performed adequately. Even when a predicted blood lead interval for a set of exposure inputs does not overlap an observed blood lead level, there may be plausible explanations owing to the complex nature of multi-media exposures and the difficulty in characterizing all the relevant determinants of these exposures, and the degree of inter-individual variation in blood lead concentrations that is known to exist even when exposure is very well characterized.

4.4.2 Data Quality

The quality of blood lead data can be specified by Data Quality Objectives (DQOs) which are established prior to the data collection effort. This DQO effort, as outlined in the Guidance for Data Useability in Risk Assessment, Part A (U.S. Environmental Protection Agency, 1992b), should result in a sampling and analysis plan which details the chosen sampling and analysis options, and provides goals for confidence intervals. The data quality indicators of completeness, comparability, representativeness, precision, and accuracy can provide quantitative measures of data quality of both sampling and analysis for blood leads concentrations.

The data quality indicators for sample collection and analysis are presented in detail in the Centers for Disease Control and Prevention protocols for blood collection and analysis. Those protocols also cover the elements of QA/QC for specimen collection, specimen preservation and shipping, analytical method performance, bench and blind quality control material, and data integrity. The following guidance is given by CDC on selecting a proficient laboratory and interpreting the results from that laboratory (Centers for Disease Control and Prevention, 1991):

> "Laboratories where blood is tested for lead levels should be successful participants in a blood lead proficiency testing program, such as the program conducted jointly by CDC, the Health Resources and Services Administration, and the University of Wisconsin. In interpreting laboratory results, it should be recognized that a proficient laboratory should measure blood lead levels to within several μg/dL of the true value (for example, within 4 or 6 μg/dL of a target value). The blood lead level reported by a laboratory, therefore may be several μg/dL higher or lower than the actual blood lead level."

In terms of evaluating the design of a sampling plan for blood lead, perhaps the most important data quality indicator is that of representativeness. Representativeness is the extent to which the data defines the true risk to human health for the population living at that site. For consideration in the risk assessment process, the sampling must adequately represent each exposure area and exposure scenario. Sampling that is nonrepresentative increases the potential for false negative or false positive results. A statistically based sampling plan is needed in order to achieve representativeness. Most studies have tried to include all children less than 84 months of age, or a random subsample of that age group. A substantial non-response rate, or attrition rate in a longitudinal study, will undermine the reliability of the

study findings. Opportunistic or selective samplings may occur with a medical referral program, a daycare center recruitment, or a community-wide request for volunteer participation, and are likely to be non-representative for the whole population. Studies in which respondent families are identified by telephone may miss families without phones, that may include transient populations and poorer populations who are possibly at greater risk.

4.4.3 Age of the Population Tested

The IEUBK Model contains uptake parameters and pharmacokinetic algorithms for children younger than 84 months of age, and predicts blood lead levels only for those ages. Infants and children younger than 84 months of age, that is, 6 months to 7 years old, have been identified as the subpopulation most susceptible to the adverse effects of exposure to low levels of lead (U.S. Environmental Protection Agency, 1986). For this reason, the blood lead study data that are to be evaluated in conjunction with the results of the IEUBK Model should consist only of those children younger than 84 months of age. If age groups older than 84 months are included in the study, it will be necessary to remove the data for these chidren from the data set, and to remove their contribution to the statistical results.

4.4.4 Time of the Year When Testing Was Done

Blood lead concentrations show seasonal fluctuations due to factors such as the relatively short half-life of lead in blood, reduced outdoor exposures in the wintertime, and perhaps to physiological (hormonal) changes. Cold weather, attending school, and snow cover tend to reduce the amount of time a child spends outdoors, and the child's direct contact with contaminated soil. The amount of this fluctuation is variable depending on physiological and behavioral factors as well as climatic ones. Seasonal fluctuations in blood lead concentrations as great as 4 to 6 μg/dL have been observed in some studies (Stark et al., 1982; Rabinowitz et al., 1984; Menton et al., 1994).

Hence, a blood lead study conducted in August would not be comparable to one conducted in March. In the 1979 to 1982 Boston lead study (Rabinowitz et al., 1984; Menton et al., 1994), blood lead concentrations associated with fluctuations in air lead and dust lead (probably from combustion of leaded gasoline) were at their maximum during the May to August period. Depending on the climatic conditions at a site, the peak summer months are an optimum time to conduct blood lead testing when soil lead is the primary source. The children are more likely to have been playing outdoors for 2 to 3 months and have had the greatest opportunity to be exposed to outdoor sources of lead.

The amount of time a child has been exposed to a specified environment is also a concern when evaluating the testing period. Because of the relatively long amount of time required for a child to come to nearly complete equilibrium with his or her environment, it is recommended that children who have lived at their current residences for less than three months or who spend more than 80% of their time away from their residences be excluded from the statistical analyses if only environmental lead data from their current residence are available. Blood lead results for these children may not be representative of the true health risk at the current residential site.

Because there are few data to quantify the impact of seasonal fluctuations on childhood blood lead, the model was calibrated using data collected during the peak summer months. Blood lead studies conducted at other times of the year should be adjusted to compensate for this seasonal difference.

4.4.5 Concurrent Characterization of Lead Sources

If a blood lead study is to be evaluated in the risk assessment process, it is important that all of the sources of lead exposure at the site be characterized and quantified. The most useful data bases contain "paired" data sets (i.e., each child's blood lead would be paired with the environmental data that represents the child's integrated exposure to lead). This pairing of environmental data with blood lead data allows the risk assessor to examine the relationship between a child's blood lead and his or her sources of exposure. At a minimum, the environmental data would include the lead concentration in soil and in house dust at the child's residence.

When the blood lead concentrations predicted by the model vary significantly from those observed in the population, this pairing of environmental and biological data provides the risk assessor with a tool by which to examine those differences. For example, were all of the children's predicted blood lead values systematically higher or lower than those observed? If so, perhaps an important source of lead exposure in the community was overlooked, perhaps assumptions about intake rates or uptake may be invalid, or perhaps unidentified behavioral variables affecting the source lead-blood lead relationship are operating. If a few individual children show particularly striking deviations of observed blood lead from predicted blood lead, then the contaminant concentrations or demographic/behavioral data for those children should be re-examined.

4.4.6 Demographics and Behavioral Factors That Affect Lead Exposure

Prior to sample collection, a well-designed blood lead study will have obtained information on the demographics and behavioral factors that affect lead exposure in a community. Such a community survey asks families about occupations, hobbies, social and economic status, house cleanliness, interior/exterior paint condition, children's mouthing behavior, etc. All of these questions are designed to identify factors that can modify the extent to which a child is exposed to the concentrations of lead in his or her environment (i.e., in media around the child). The Demographics Workplan for the California Gulch Study Area is an example of one such survey (Woodward-Clyde, 1991).

The results of the community survey can be used to evaluate differences between blood lead concentrations predicted by the model and those observed. Affirmative answers to "Have you sanded the paint in your home recently?" or "Does your child eat paint chips frequently?" may highlight why some predicted and observed levels differ. With the information from these surveys, a risk assessor can evaluate differences between observed and predicted blood concentrations due to behavioral or demographic factors.

4.4.7 Effect of Public Awareness or Educational Intervention

Whether or not a community's awareness of the hazards of lead exposure can cause its members to act to alter blood lead levels is an unresolved question. It is possible that an enhanced awareness of lead exposure in a community could prompt that community to alter behaviors to reduce lead exposure, and subsequently, reduce blood lead concentrations in that community. However, the empirical data on this phenomenon are very limited. Anecdotal evidence suggests that one-on-one counseling and educational intervention targeted specifically toward high risk children is effective in reducing individual blood lead concentrations (personal communications: R. Bornschein, 1992; I. Von Lindern, 1992). We are not aware of any study that has been designed specifically to test the effectiveness of educational intervention. A good study design is needed to avoid both statistical and sampling biases.

Whether or not a general type of awareness in a community may elicit a similar response has yet to be determined. The differential effectiveness of public awareness campaigns about soil and dust lead hazards in different subpopulations has also not been investigated. A study in Raleigh, NC, found that the greatest response to the city's offer to

test tap water for lead (at no cost to the water customer) was from the higher income neighborhoods of the city (Simmons, 1989).

Therefore, when a risk assessor is evaluating a blood lead study, he or she should keep in mind the potential effect of public awareness on blood lead concentrations. If active educational intervention and counseling programs are being conducted at a site prior to blood lead collections, or if there is a high level of citizen concern about contaminated sites, the results of that blood lead study may be different than it would have been otherwise.

4.4.8 Comparison of Observed and Predicted Blood Lead Concentrations

4.4.8.1 Were Important Sources of Lead Exposure Overlooked?

Unless site-specific data are provided by the user to the IEUBK Model for soil, house dust, air, drinking water, and diet, the model will assume a standard default value for intake from each medium. For example, at a site where the soil lead concentrations are elevated and homegrown fruits and vegetables are a large part of the diet, the diet pathway may be contributing more significantly than the model assumes to total lead exposure. The standard diet default value in the model is based on recent FDA market-basket survey information and pertains to lead concentrations in store-bought food. It doesn't consider the contribution of lead from homegrown fruits and vegetables, which may vary from site to site depending on the soil lead concentration, soil conditions, type of produce, climate, etc. Communities that have large ethnic minority populations may also have unique sources of childhood lead exposure in folk medicines or cosmetics that use lead compounds, or in foods imported in lead-soldered cans.

Ingestion of paint chips is another source of exposure that may be overlooked. Exposure to lead occurs from deteriorating house paint via ingestion of paint chips, and via ingestion of fine particles of paint in household dust. Exposure to fine particles of lead-based paint in dust and soil is handled through the soil/dust menu. For ingestion of paint chips, however, the IEUBK Model assumes a standard default of 0 µg/day for le from paint chips and other alternate sources.

In addition to examining the possibility of overlooking an important source of lead exposure, the risk assessor should examine the representativeness and accuracy of the environmental data that were collected. For example, is the model input value for lead concentration in drinking water based on first draw tap samples, groundwater samples, or estimates from public water company records? A weighted combination of first draw and flushed tap water samples (plus water from school or day care fountains, if applicable)

provides the most appropriate representation of the average lead values in a child's drinking water. The farther away you move from these sources of information, the less accurate and more uncertain your input to the model will be.

Is the soil lead input based on the average of soil lead concentrations over the entire yard or is it based on composite samples from a child's yard? If there is substantial variability in soil concentrations at different locations about the yard, as is often true, an average of the entire yard may not be an accurate estimate of risk. An integrated assessment using the perimeter, play areas, and bare areas from each child's residence would provide an alternative basis for estimation.

Ideally, the inputs to the model should represent the integrated daily exposures each child might be expected to have. The absence of data specifically collected to estimate the integrated exposure will limit the accuracy of an analysis. Refining the accuracy and representativeness of the environmental data values provided to the model may be useful in resolving differences noted between estimated and observed blood lead concentrations.

4.4.8.2 Are There Interrupted or Enhanced Exposure Pathways at the Site?

A mistake that is often made is equating contaminant concentration with exposure or risk, where the risk assessor assumes *potential* exposure is *actual* exposure. Briefly, if there is no exposure, there is no risk. If an exposure pathway is diminished or enhanced, then regardless of contaminant concentration, the resulting exposure or risk is also diminished or enhanced. For example, at the same concentration of lead in soil, exposure to bare soil may be greater than if the soil has a good vegetation cover.

4.4.8.3 Are the Assumptions About Site-Specific Intake Rates and Uptake Parameters Valid?

Internal (systemic) exposure for humans is a function of contaminant concentration, intake rate and uptake. Environmental sampling can be designed and conducted to obtain a reasonably accurate representation of the lead exposures a child might experience at a site, thereby reducing some of the uncertainty in the exposure estimate. However, it is more difficult to reduce the uncertainty about the site-specific intake rates (i.e., soil ingestion rate, water ingestion rate) and uptake parameters.

At this time, the empirical evidence on these assumptions is limited and variable. In other words, there is a degree of imprecision and uncertainty in the intake rates and uptake parameters. For example, bioavailability of lead from soil is one uptake parameter to which the model is very sensitive. The model assumes a standard default of 30% for

absorption of lead from soil in the gastrointestinal tract, yet existing bioavailability studies in animals show values ranging from 5 to 40%. Concerns exist about the design of, and animal models used in, these studies (Section 4.1). Site-specific adjustments in the uptake parameters require strong justification.

A risk assessor should first explore all of the other rationales for differences between observed and predicted blood lead concentrations (i.e., sources of lead that were overlooked, incorrect assumptions about pathways, inaccurate estimates of environmental intake, and inadequate information about important or relevant demographic/behavioral factors). The risk assessor should then have strong site-specific justification before exploring non-default assumptions about uptake parameters.

4.5 ASSESSING THE RELATIONSHIP BETWEEN SOIL/DUST AND BLOOD LEAD

4.5.1 Assessing Reductions in Blood Lead

The IEUBK model can be used to estimate the change in geometric mean blood lead from reducing lead exposure, provided the exposure has remained stable for at least three months and there is a sufficiently detailed characterization of post-reduction lead exposure. This means that it is necessary to calculate the post-reduction levels for the controlled medium, the recontamination of the controlled medium by sources of lead exposure that are left after reduction, changes in the other exposure media from different pathways, and changes in physical or chemical properties of all media that may affect access, intake, and bioavailability to children.

There are not many data on post-abatement environmental lead concentrations for nonurban sites, such as smelters or lead mining sites. As an example, suppose that a primary lead smelter has been closed down. This immediately reduces or eliminates air-borne leaded particulates. Over the next few months, fine surface particles in household dust not otherwise trapped by carpets, upholstered furniture or inaccessible nooks and crannies, will be gradually swept, washed, or blown out of the house. If replaced by new surface soil particles, these will be much lower in lead than before the smelter was shut down, so that the household dust lead concentration may be expected to decrease within characteristic time scales of a few months to a new quasi-equilibrium value. The surface soil that had high concentrations of lead before the smelter was shut down may gradually be worn

away by wind or water erosion, but over a period of many years. This pattern is an informa- description of what has actually been observed at the Bunker Hill site in northern Idaho.

The IEUBK model may be used with long-term post-abatement values to predict blood lead concentrations in children occupying these residences long after abatement has been carried out, without worrying about the dynamics of soil and dust lead changes over time. However, the post-abatement soil and dust at a specific site may not be the same as pre-abatement soil and dust at the same site. If highly aggregated soil is replaced by loosely consolidated fine particles in clean fill soil, and is not adequately covered by grass or sod, then the post-abatement soil may be both more easily transported into the house and more bioavailable than before abatement. Conversely, if the grass or sod cover is maintained well after abatement, then the post-abatement soil-to-dust lead coefficient in the IEUBK model may be different than the pre-abatement value. The validity of the IEUBK model predictions for post-abatement risks is limited by the validity of the input parameter assumptions for post-abatement exposures.

At present the definition of elevated blood lead (EBL) is the level of concern of 10 μg/dL defined by USEPA (1990b) as the lower limit of the range of known possible adverse neurobehavioral effects in young children. The protection level most often used in practice is a maximum 5 percent risk of elevated blood lead (EBL) for children in a given household.

The user has the responsibility for using model input parameters that are appropriate to the site. Collecting an adequate number of representative soil and dust samples, and determining their lead concentrations and physical or chemical properties that affect transport and bioavailability, are generally the minimum site-specific data collection and analyses that are needed. The ideal input data includes (1) a multimedia household environmental lead study that includes soil, dust, paint, water and air; (2) information on lead exposures outside the child's home; and (3) information on family demographics and child behavior patterns in the community that may affect access to lead sources; (4) characterization of physical and chemical properties that affect bioaccessibility and bioavailability.

Interest has been growing in the potential uses of the IEUBK model for sites at which there is presently no residential housing, or at sites at which children may be exposed without residential dwelling units being physically on the site. Since the IEUBK model calculates expected geometric mean blood lead concentrations and EBL risks for hypothetical populations of children, the model can be used for these applications. This can be done only

if there is sufficient information on child exposure to estimate time-weighted or activity-weighted soil lead and dust lead concentrations, combining both residential and on-site exposures.

4.5.2 Situations in Which the Use of the Integrated Exposure Uptake Biokinetic Model Is Uncertain

4.5.2.1 Assessment of Risk with Community or Neighborhood-Scale Input

There are situations in which it is either inconvenient or impossible to apply the IEUBK model at the intended household residence scale. For example, only mean values or geometric mean values of input parameters such as soil and dust lead may be available for a group of households. Another possibility is that there are a substantial number of soil and dust lead measurements at a site, but not at houses or locations within the site where blood lead and EBL risk estimates are needed. We have little information on applications of the IEUBK model with larger-scale input data, and we must caution the user against using the IEUBK model for this purpose, because little is known about blood lead variability in such situations.

4.5.2.2 Use of Surrogate Input Data from Models or Surveys

When modeled or survey data is to be used as input in the Lead Model, the user should consider the collection time and scale of the data in order to obtain maximum predictability in the output. Applicability to the individual home, neighborhood area or community should also be demonstrated. For example, housing age can provide a useful screening variable for field measurements of lead in tap water and lead-based paint, but it is not likely to be an adequate substitute for the lead concentration data unless a quantitative predictive relationship can be established by other studies in the same home, neighborhood or community. Such screening variables may be useful in screening for areas of concern for lead exposure sources. At the same time, the output values should not be construed as accurate representations of the actual child blood lead levels in these areas.

4.5.2.3 Use of the Model To Assess Risk of Elevated Blood Lead at the Regional or State Level

There is no empirical basis whatever for using the present version of the IEUBK model at this scale. We have serious concerns that large-scale input data may be totally inadequate characterizations of the spatially confined exposure for any individual child.

4.5.2.4 Use of the Model To Assess Trigger Levels for Soil Abatement at the Community, Regional, or State Level

Use of the present version of the IEUBK model at this scale is discouraged, because risks cannot be estimated adequately.

4.5.3 Factors That Constrain or Limit the Use of the Model

4.5.3.1 Data and Data Sets Used as Input for the Integrated Exposure Uptake Biokinetic Model

Residential Versus Commercial/Industrial Sites

The IEUBK Model uses site-specific data on the lead concentrations in air, water, soil and household dust, and average daily intake of lead from diet and from direct ingestion of paint chips, to estimate the geometric mean blood lead in children exposed to environmental sources of lead. The data input requirements assume a residential exposure, and thus the output of the IEUBK Model with default assumptions is probably not predictive for industrial or commercial sites at which exposures for small children are restricted, except perhaps in assessment of future use scenarios, or as additive components to a residential exposure scenario. Development of model estimates in such situations would require adequate specification of soil and dust ingestion derived from the contaminated site.

Age Group for Which Data Is Available

The IEUBK Model contains data and algorithms to determine intake, absorption, excretion and movement of lead between body pools for children from 6 months to 7 years of age. The IEUBK Model is only predictive for children in this specified age range or any subinterval within this range. Future versions of the IEUBK Model may be expanded to include data on metabolic processes in older children and adults, and thus allow characterization of blood lead levels in these populations.

At present, the IEUBK Model cannot be used to characterize blood lead levels in children older than seven years or in adult populations.

Other Critical Subpopulations

The IEUBK Model does not predict the blood lead levels of *pregnant women*, given either default or site-specific exposures. A parameter input for the maternal blood lead level has been provided in the IEUBK Model to capture the effect of prenatal exposure in unusual circumstances of exposure, i.e., in occupational settings. In general, maternal lead exposure during pregnancy is not well characterized for changes that occur from pre-pregnancy baseline. The adverse effect of prenatal lead exposure on neurobehavioral and physical

development is highly significant, and future versions of the IEUBK Model may include a prenatal exposure component based on the transfer of lead from the mother's blood to the fetus at the time of birth.

The IEUBK Model contains no specific data to differentiate the adverse effects of lead on different racial or ethnic groups, nor is there sufficient published data to develop this component. However, exposure scenarios for a specific subpopulation may be provided by the user if data are available.

Residency Requirements

The IEUBK Model does not allow entering rapid time-varying changes in exposures to lead sources. The IEUBK Model has been developed using blood lead data from children who have had at least a three-month exposure to their residential sources prior to blood sampling for lead analysis, that is, a minimum three-month residential requirement for inclusion in blood lead studies. The three month residency requirement guarantees that predicted blood lead attributable to the current residential exposure will be nearly at a steady state level. If residency requirements have not been met or if lead exposures are changing rapidly, the IEUBK Model can be expected to give less than accurate predictions, because exposures at prior residences may still be a major determinant of blood lead.

Timing of Data Collections

Because of the variability in child blood lead levels with seasonal exposure and the corresponding variability in environmental lead levels (i.e., changes in household dust lead levels with seasonal and activity changes) strict attention should be paid to the timing of data collections if the data is to be used as input in the IEUBK Model to make predictions about individual or community blood lead levels in children. This is especially important if the predicted blood lead levels are to be compared with the results from a community blood lead study, to assure that the two studies measure the same population at the same period in time. same season of the year. The parameters for the IEUBK model were developed from diverse animal and human studies. Collectively, these studies reported ranges of values for these parameters. The first stage in model validation was a calibration stage, using paired data—measurements of lead in environmental media and in blood collected from children under the age of six, taken within a short period. Comparison of observed and predicted blood levels suggested modifications of the parameters, within the range of plausible values suggested by the literature or by our analyses of research data. After there adjustments, the model obviously could not appropriately be tested again using the same set of data. Therefore,

validation tests were performed using the sets of community blood lead data paired with environmental exposure data for the same child.

4.5.3.2 Biological and Exposure Parameters Used in the Integrated Exposure Uptake Biokinetic Model Bioavailability of Soil Lead

The bioavailability of lead from different sources may be variable due to differences in lead concentration, lead speciation, particle size and mineral matrix (Barltrop and Meck, 1975; Barltrop and Meck, 1979; Heard and Chamberlain, 1982; Rabinowitz et al., 1980; Cotter-Howells and Thornton, 1991; Aungst and Fung, 1981). Additionally, bioavailability may vary as a function of physiological parameters such as age, nutritional status, gastric pH, and transit time. The IEUBK Model uses a default of 30 percent lead absorption from soil, which is constant across all concentrations and soil sources. Site-specific data on the soil and dust bioavailability may improve the accuracy of the blood lead level predictions.

Other Lead Exposure Inputs

Child default values for dietary lead intake are provided by year and by age of the child in the IEUBK Model. The use of default values is appropriate unless the dietary lead intake is very high, due perhaps to a high intake of home-grown fruits and vegetables or the intake of lead-contaminated ethnic food or drugs.

Exterior lead-based paint can make a significant contribution to soil lead, and is usually considered as part of this exposure source. The contribution of lead-based paint to indoor household dust is harder to estimate because the condition of the paint varies from house-to-house and the rate of incorporation into house dust is variable. If the household lead-based paint contribution is highly variable in a community, care should be taken to avoid combining all homes in a single run of the IEUBK Model, as the output results may not be applicable to the population.

Children can eat chips or strips of deteriorating lead-based paint directly from painted surfaces, even when the total area of lead-painted surfaces is so small that the total contribution of lead-based paint to interior household dust or exterior soil is too small to identify. Paint chip intake reflects child-specific behavior, including observed ingestion of paint chips, observed contact of the child's mouth with painted surfaces and the frequency of mouthing of non-food objects.

Blood Lead Variability

The variability of individual blood lead levels with respect to the geometric mean blood lead level predicted by the IEUBK Model is characterized by a single number: the geometric

standard deviation. The GSD is used as a single number to characterize the relative variability of the log-normal distribution representing the aggregate uncertainty in all sources of population variability: biological, uptake, exposure, sampling and analytical.

A common misconception is that the IEUBK Model predicts the community geometric mean blood lead and the fraction of children at risk when the input is the arithmetic mean or geometric mean across households of household-specific lead concentrations. This use of the IEUBK Model may cause seriously misleading interpretations of the output of the model, when the true extent of variability is not known. A correct approach to neighborhood risk estimation is given in Section 4.2.5.

Prior Body Burden

Child blood lead level predictions obtained using the IEUBK Model reflect the contributions from lead sources entered into the model; they do not take into account any existing body burden which may be the result of prior exposures not known to the user. Current blood lead levels depend on prior exposure history as well as present exposure. If past exposure levels have been greatly elevated, the results obtained from the IEUBK Model may not be accurate. Where children have had high prior exposures, that prior exposure affects blood lead levels for at least three months after the exposure ends, a "washout" period. Future estimates are based on present conditions. If those conditions change (e.g., deteriorating paint that might change house dust lead concentrations), the exposure and consequent risk will be different.

Alternate Exposure Locations

Child blood lead levels obtained using the IEUBK Model refect input lead sources at the household level or neighborhood level. They do not necessarily take into account increased or reduced lead exposures which may have taken place at parks, preschool, homes of babysitters, neighbors or relatives, or other locations frequented by the child, unless these exposures are measured and explicitly entered into the model as inputs. Thus, the results obtained from the IEUBK Model may not be accurate unless the child's activity patterns have been well documented.

Socioeconomic Status

The blood lead levels of two children with identical lead exposure scenarios, but living in different family behavior patterns might vary greatly. The difference in socioeconomic status might be reflected in differences in household repair and cleaning, washing of children's hands and toys, food preparation methods, concern for balanced meals and

improved nutritional status, more regular eating patterns, etc., all of which may impact blood lead levels. Use of the IEUBK Model should be preceeded by adequate characterization of information on behavioral and other socioeconomic differences, and advice from regional offices on appropriate adjustments, if warranted.

Intervention/Public Education Programs

Intervention and public education programs can inform the community of the adverse health effects of lead exposures and how to reduce them. These activities may result in reductions in blood lead levels in portions of a community that may be temporary, depending on how well the information is conveyed and received. These temporary changes in blood lead concentrations might occur during a one-time blood lead survey and cannot be predicted using the IEUBK Model. Some of the examples in Chapter 5 describe the correct application of the model in this situation.

4.6 WHAT YOU NEED TO KNOW ABOUT BIOKINETICS

4.6.1 Description of the Biokinetic Model

The IEUBK model has a very detailed biokinetic modelling component. This component of the model is not accessible to the user because, in our judgement, most users will neither wish to change the biokinetic parameters nor have the need to change any of the biokinetic parameters. The biokinetic parameters are used to define intrinsic biological variables that do not change from one exposure scenario to another, once a child's age is specified. The basis for the biokinetic parameters are described in the Technical Support Document: Parameters and Equations Used in the IEUBK Model for Lead in Children (see Section 1.2.2).

The biokinetic model is a compartmental model, in that it assumes that all of the lead in the child's body can be attributed to one of seven kinetically homogeneous compartments and that transfer between these compartments occurs through normal physiological processes. The compartments in this model are:

Plasma and extra-vascular or extra-cellular fluids (denoted ECF);
Red blood cells
Kidney
Liver
Other soft tissues

Trabecular (spongy) bone
Cortical (compact) bone

The distribution of lead in the body is only approximated by a compartmental structure, even in so-called physiologically-based pharmacokinetic models, because no tissue compartment is, in reality, completely homogeneous. However, the compartmental method is so useful and accurate that it has been almost universally adopted.

Realistic growth equations are used for each organ or tissue pool (biokinetic compartment) from newborn status to age 7 years. The transfer times (equivalently, fractional transfer rates) among compartments are scaled according to organ volume or weight, or body volume or weight, using allometric scaling consistent with organ or body surface-area scaling. The basis for the compartmental transfer times are the reanalyses we have done for data from studies in infant and juvenile baboons, using data in (Mallon 1983) and (Harley and Kneip 1985). A wide variety of studies in human children and adults, in other species, and in other metals was used to estimate biokinetic parameters not estimatable from the baboon studies. Growth equations were derived from Altman and Dittmer (1962), Spector (1956), and Harley and Kneip (1984). The literature review revealed 17 adult and 3 pediatric studies for evaluating the transfer time from blood to urine. An allometric scaling factor, based on the correlation between body surface area and glomerular filtration rate (West, 1948), was applied to the transfer time composited from the 17 adult studies to provide an estimate of the blood to urine transfer in children. An estimate of transfer from blood to feces and blood to urine for adults was taken from Chamberlain et al. (1978) and Rabinowitz et al. (1976), and for transfer from blood to soft tissues from Rabinowitz (1976), and equations for compartment to blood lead concentration ratios from Barry (1981).

The flow of lead from external media into the body and the distribution and elimination of lead is shown graphically in Figure 4-10. Transfer of lead to and from plasma and extravascular fluids is governed by first-order kinetics, in that the rate of change of the lead content in each compartment is a functi . of the current state of the system as defined by the lead content of each of the compartments. If the dependence of the rate of change of lead content is a linear function of the contents of all of the compartments, then the biokinetic model is described as a first-order linear kinetic model. The IEUBK has almost linear kinetics, except that we assume that the lead-binding capacity of the red blood cells can be saturated when lead uptake into the body is very high. Uptake of lead can occur through the lungs into the plasma-ECF pool, or through the gut into the plasma-ECF pool. While the

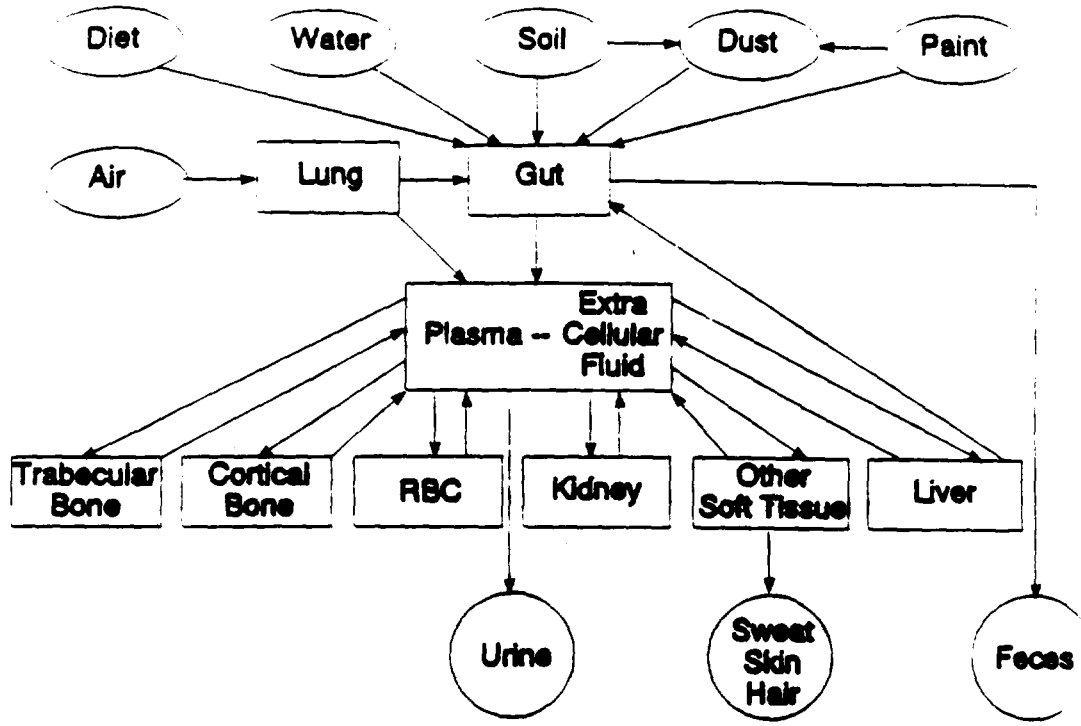


Figure 4-10. Biokinetic compartments, compartmental lead flows, and uptake pathways in the integrated exposure uptake biokinetic model.

plasma-ECF pool may be viewed as the central pool or compartment in this system, the usual observable is the blood lead concentration, which combines both the lead in plasma-ECF pool and the lead in the red blood cells.

4.6.2 Consequences of Biokinetic Para...eters for Site-Specific Risk Assessment

The exposure scenarios that can be used in the IEUBK model change only once a year. Since most of the transfer times for children are on the order of 1 hour to 1 month, the IEUBK calculations of lead levels in blood and soft tissues may be assumed to be in a quasi-steady state condition with respect to exposure. The quasi-steady-state condition may allow the use of simple linear approximations to blood lead vs. media concentration or media intake of lead at different ages. But the time scale for release of lead from bone is much

longer, on the order of 1 to 3 years, so that the bone lead level quickly builds up and the skeleton contains 60 to 70 percent of the total body burden of lead by age 2 years. There is not be a true steady-state blood lead level during the 7-year interval used in the IEUBK model for young children. As bone lead burdens increase, so will there be a growing component of lead in blood that comes from release of lead from the skeleton or from resorption of bone in growing children.

Because of this release of bone lead, there may be a large component of blood lead levels in children that will respond only slowly to any changes in environmental lead exposure. This is particularly noticeable in evaluating soil lead abatement studies and strategies, where a child may have accumulated a large body burden of lead before the abatement. In the first year or two after the abatement, the internal or endogenous source oflead stored in the skeleton may cause a moderately elevated blood lead level to persist in the child. Children who were never exposed to the elevated environmental lead, or who did not accumulate a large body burden of lead before the abatement even though the environmental exposure was high, will not have this residual elevation of blood lead from resorbed bone lead.

4.7 ISSUES IN USE OF THE MODEL FOR PAINT CHIPS

4.7.1 Inappropriateness of Use of IEUBK Model for Paint Chip Ingestion

The IEUBK model, Version 1.0, does not contain an explicit component for lead-based paint ingestion outside of the Alternate Source Option in the Soil/Dust Menu. The correct use of the IEUBK model is to estimate geometric mean blood lead levels and distributions of blood lead levels in young children who have long-term chronic exposures to lead. It has long been known that the ingestion of even tiny quantities of paint chips on a single occasion can cause serious lead intoxication. Chisolm and Harrison (1956) show photographs of small paint chips weighing several grams that can easily be removed and eaten by a child. Since old lead-based paints can contain in excess of 50 percent lead, the child may ingest several million micrograms of lead in a single episode. The IEUBK model is not intended to address this situation. The IEUBK model is intended to address the situation where the child ingests typical quantities of household dust that have been contaminated by leaded soils and by deterioration of old lead-based paint from interior surfaces. The inclusion of lead-based paint in the dust menu implicitly assumes that paint has fallen off the painted surface as fine particles, or has fallen off as discrete flakes or chips of paint and has been reduced to small

particles in situ on the floor, carpet, furniture or other surfaces. Interior lead-based paint may not wear as rapidly as exterior paint due to the near-absence of sunlight on most household surfaces, but common observation finds many deteriorated lead-painted interior surfaces in older housing, especially in wet rooms such as kitchens, bathrooms, or laundry rooms (HUD, 1991).

The following data are presented to assist the user who wishes to develop an exposure scenario in which there is long-term ingestion of chips of lead-based paint, in addition to the interior household dust lead contribution that is already included in the IEUBK model. An exposure scenario with paint chip ingestion can be entered in the Other Source Menu of the model. The data for construction of an alternative lead-based paint chip menu were reviewed by the EPA Technical Review Work Group, who concluded that these data were not adequate to be recommended as default values. There are greater uncertainties about paint chip exposure and uptake than about other exposure media. These uncertainties include:

(1) The quantity of paint chips ingested on a long-term or chronic basis is unknown; however, even small quantities of ingested paint chips can produce a lead intake of millions of micrograms per day, overwhelming all other sources.

(2) Lead levels in housing are most typically measured as surface loadings using portable XRF analyzers. While there are several proposed relationships between lead paint surface loading and daily lead intake, these require making assumptions about other uncertain relationships, such as the "area" of surface ingested the child, or the thickness of the paint chip and the relationship between lead concentration and lead loading. We will describe these relationships, but we believe that they do not yet have an adequate empirical basis.

(3) Paint chips are, by definition, discrete units. Even if paints chips are at least one millimeter in diameter, or even larger, they may not be completely dissolved in the stomach or completely absorbed in the intestines. Observations of child fecal samples sometimes find discrete paint chips. Radio-opaque samples in stool may be lead or some mixture of lead with other heavy metals such as barium or chromium commonly found in leaded paint pigments.

(4) Lead paint absorption by rats has been found to depend significantly on particle size and chemical speciation of paint particles. Many chemical species are found in lead-based paint, most often including lead octoate (as a dryer), lead carbonate, and lead chromate. The sequence of absorption or bioavailability is probably

$$\text{carbonate} \geq \text{octoate} > \text{chromate}$$

based on rodent studies (Barltrop and Meek, 1979). While the ranking is probably similar in human children and other primates , direct evidence is limited to baboons (Cohen, 1975; Mallon, 1983). Studies are currently in progress using miniature swine as closely analogous models of human gastro-intestinal absorption of nutrients and contaminants, but results on absorption of lead from actual lead paints have not yet been reported. It is clear in any case that estimates of lead bioavailability in paints may require a much more complete site-specific characterization by particle size and chemical speciation than does soil.

4.7.2 Daily Intake of Paint Chips

The American Academy of Pediatrics (1972) has used a provisional estimate of one square inch (6.25 cm^2) of paint surface ingested per day. This appears to be a nominal value for purposes of risk estimation, and no empirical basis for this value has been provided. They cite evidence that 1 cm^2 of one layer of interior paint may weigh 5.0 to 8.2 mg (average 6.5 mg), and that six layers of paint weighed 37.0 to 40.6 mg (average 38.8). Thus, using data that may represent Providence RI in 1972, where six layers of paint were typical, ingestion of 6.25 cm^2 of painted surface through a single painted layer would correspond to 40.6 mg/day intake, and a thick chip containing six layers would average 233 mg/d paint chip intake. Even if the ingested paint chips were square-inch monolayers with one percent lead, the daily lead intake would be 400 ug Pb/d. We cannot provide a realistic estimate of the uncertainty of this estimate. It is likely that there is some correlation between the size, thickness, and lead content of ingested paint chips, since additional lead is reported to add a sweet taste to the chips that may appeal to a child with pica for lead paint chips.

These estimates were also cited in a report by the National Academy of Sciences (NAS, 1973) to the Consumer Product Safety Commission (CPSC). They concluded that the

quantitative evidence was inadequate "to promulgate a standard based on knowledge of the essential quantitative relations that link the lead content of paint to symptoms of intoxication. However, this is not unusual in public-health practice. Many useful standards have been established by informed people who make judgments based on whatever facts are available" (NAS, 1973, pp. 25-26).

In view of the lower quality of information on paint chip intake than on intake of soil and dust, diet, and drinking water, and the usefulness of providing baseline risk assessments in the absence of lead-based paint, we have used a default value of 0 μg/dL in the model.

4.7.3 Relationship of XRF Lead Paint Surface Loading to Lead Paint Concentration

The estimate of lead intake from paint chip ingestion depends on a lead concentration for the ingested chips. However, this is not available in field samples without removing a piece of paint from the wall or trim. Therefore, the use of non-destructive field sampling methods such as portable XRF analyzers has become the common method for determining paint hazard. We can calculate

$$\text{lead concentration } (\mu g/g) = 0.001\ (\mu g/mg) * \text{lead loading } (mg/cm^2) /$$

$$\text{thickness of paint (cm)} * \text{paint density } (g/cm^3).$$

Calculations from the EPA Lead Reference Materials Workshop (EPA 1991) assuming a seven-layer thickness of paint (40 mil = 1 mm) and a density of 2 g/cm^3 calculates 5,000 $\mu g/g$ equivalent to 1 mg/cm^2. This is reasonably concordant with some analyses of measurements of paint loading and concentration that we had calculated from data in the Boston Brigham and Women's Hospital Longitudinal Lead Study. However, this relationship is likely to vary so greatly from house to house that we cannot recommend its use without site-specific verification.

4.7.4 Dissolution of Paint Chips in Acid Environments

Not all of the lead in a large lead paint chip may be available for absorption. Roberts et al. (1974) report that "20 to 60 percent of the lead in surface soil was extractable in 0.1N HCl compared with less than 10 percent extractable from paint samples." Particle dissolution is a component of lead bioavailability.

4.7.5 Absorption of Lead Paint In Vivo

The absorption of lead-based paint particles by rats is described in (Barltrop and Meek 1979). They conclude that "The physical form of particles derived from paint film would seem to modify the availability of Pb compounds contained in them for absorption. Little is known of the physical or chemical changes which paint flakes undergo after ingestion, although it is known that some paint flakes remain relatively intact when swallowed by a child and may be observed radiographically in the gut lumen, or on inspection of feces. In spite of this, sufficient absorption of Pb resulting in childhood poisoning is known to occur, and in many cases the ingested flakes become too finely divided to be visible macroscopically. Thus the composition of the paint and the chemical nature of the added Pb compounds may determine its stability in the gut and hence the availability of Pb for absorption. Long-term feeding of paint flakes identical to those used in this work, but of larger size (500 to 1,000 microns) have been reported to result in minimal absorption by the rat (Barltrop and Meek, 1975)."

Table 4-4 summarizes their results. Lead absorption can be characterized by the difference in blood lead levels between exposed and control rats. The increase in blood lead for rats fed lead octoate in particles between 500 and 1,000 microns diameter is about 60 percent of the absorption of lead octoate particles <50 microns, and absorption of lead chromate paint in particles of 500 to 1,000 microns is about 45 percent of the absorption of lead chromate paint in particles of 500 to 1,000 microns. For particles <50 microns, the increase in blood lead for lead octoate particles is about 60 percent of the increase from lead acetate. It is not clear how these results can be used quantitatively for humans to determine absolute or relative bioavailability of LBP.

Juvenile and infant baboons were exposed to oral intakes of lead salts and prepared lead paint samples from New York City (Mallon, 1983). The lead salts and paint samples were fed in gelatin capsules to sedated baboons. The relative bioavailability could be estimated from differences in the steady-state blood lead levels achieved after 5 or 6 months of chronic exposure. These are shown in Tables 4-5 and 4-6. The increase in blood lead in infant baboons (age 6 months at the start of the study) was 23 μg/dL (no s.e.) for 2 baboons exposed to lead acetate and 6.125 μg/dL for 8 baboons exposed to New York city paint at a controlled dose of 100 μg/kg/day (roughly 250 to 350 μg/day in baboons who grew from 2.5 to 3.5 kg body weight). At higher doses, the increases in blood lead were clearly nonlinear with respect to dose rate. In juvenile baboons (ages 20-24 months at the beginning of the study) the increase in blood lead was 11.7 μg/dL (no s.e.) for 2 baboons exposed to

TABLE 4-4. PERCENTAGE INCREASE IN BLOOD LEAD LEVELS IN INFANT MALE WISTAR RATS WITH 48-HOUR ORAL EXPOSURE TO LEAD ACETATE, AND TO LEAD OCTOATE AND LEAD CHROMATE PAINTS OF DIFFERENT PARTICLE SIZES

Paint Chip Size (mm)	Lead	Dose Rate µg/kg/d	Blood Lead (S.E.) µg/dL	Blood Lead - Control µg/dL	Percent of PbAc
-	CONTROL	0	8.1 (1.9)	-	-
-	ACETATE	33000[1]	38.3 (4.0)	30.2 (4.4)	-
0.5-1	OCTOATE PAINT	33000[1]	19.3 (3.7)	11.2 (4.2)	37.1
<0.05	OCTOATE PAINT	33000[1]	27.2 (4.0)	19.1 (4.4)	63.2
0.5-1	CHROMATE PAINT	33000[1]	14.5 (3.2)	6.4 (3.7)	21.1
<0.05	CHROMATE PAINT	33000[1]	22.8 (2.2)	14.7 (2.9)	48.7

[1]Calculated as 0.02% lead in diet, per 31 to 33 g diet in 48 h, per 96 g body weight (range 90 to 103 g).

Source: Adapted from Baltrop and Meek (1979).

TABLE 4-5. PERCENTAGE INCREASE IN BLOOD LEAD LEVELS IN INFANT BABOONS WITH CHRONIC EXPOSURE TO LEAD PAINT, LEAD ACETATE, AND OTHER LEAD COMPOUNDS

Age	Lead	Dose Rate µg/kg/d	Blood Lead (N) µg/dL	Blood Lead - Ctrl. µg/dL	Percent of PbAc
5-6 mo	CONTROL	0	9 (1)	-	-
	ACETATE	100	32 (2)	23	-
	ACETATE	200	42 (2)	33	-
	ACETATE	1000	72 (1)	63	-
	CARBONATE	1000	69 (1)	60	95.2
	OCTOATE	100	90 (1)	81	352
	PAINT	100	15.12 (8)	6.12	26.6

Source: Adapted from Mallon (1983).

lead acetate and 33.7 µg/dL for 1 baboon exposed to lead octoate at 100 µg/kg/d, but only 3.7 µg/dL (no s.e.) in 2 baboons exposed to New York city paint at a controlled dose of 200 µg/kg/day. The increase in blood lead was 31.7 µg/dL (no s.e.) for 2 baboons exposed to lead acetate and 93.7 µg/dL for 1 baboon exposed to lead octoate at 500 µg/kg/d, but only 12.7 µg/dL in 2 baboons exposed to New York city paint at a controlled dose of 500 µg/kg/day. Therefore, the bioavailability of lead in actual paint samples was at most

TABLE 4-6. PERCENTAGE INCREASE IN BLOOD LEAD LEVELS IN JUVENILE BABOONS WITH CHRONIC EXPOSURE TO LEAD PAINT, LEAD ACETATE, AND OTHER LEAD COMPOUNDS

Age	Lead	Dose Rate μg/kg/d	Blood Lead (N) μg/dL	Blood Lead - Ctrl. μg/dL	Percent of PbAc
20-24 mo	CONTROL	0	12.33 (3)	-	-
	ACETATE	100	24 (2)	11.67	-
	ACETATE	500	44 (2)	31.67	-
	OCTOATE	100	46 (1)	33.67	288.6
	OCTOATE	500	106 (1)	93.67	295.8
	PAINT	200	16 (2)	3.67	31.4[1]
	PAINT	500	25 (1)	12.67	40.0

[1]Calculated relative to 100 μg/kg/d lead acetate.

Source: Adapted from Mallon (1983).

25 to 40 percent of the bioavailability of lead acetate administered during chronic exposure studies at dose rates roughly comparable to those assumed in the American Academy of Pediatrics report. The much higher relative bioavailability of the pure lead octoate compound remains to be explained. The absolute bioavailability of lead acetate in diet estimated by Mallon was estimated by Mallon was 24 percent at a dose rate of 12 μg/kg/d, 8 percent at 100 μg/kg/d, and 6 percent at 200 μg/kg/d in infant baboons; 12 percent at 12 μg/kg/d, 3 percent at 100 μg/kg/d, and 1 percent at 1,000 μg/kg/d in juvenile baboons. The estimates of absolute bioavailability of oral lead acetate developed by Marcus (1992) using a saturable absorption mechanism to account for the bioavailability were higher, about 28 percent and 20 percent at dose rates that were much less than 200 μg/kg/day. The bioavailability of these lead-based paints must then be taken as less than 7 percent and 5 percent respectively. A detailed characterization of the chemical composition and size distribution of the prepared paint samples would have been useful, but was not presented.

5. APPLICATIONS WITH EXAMPLES

5.1 APPLICATIONS FOR POPULATION ESTIMATES

The purpose of this chapter is to provide concrete examples complete with explanations that can guide the user through specific applications of the model. These examples are taken in part from past applications of the model, but they have been modified for the purposes of illustration and do not represent any specific site or risk management decision. While the user should find some guidance in these examples, they are not meant to be comprehensive of all possible model applications, nor should they be generalized to any particular site.

EXAMPLE 5-1. Default Values

As stated earlier in this manual, the model can predict geometric mean blood lead levels in a population of children with residential and neighborhood exposures, provided that the distribution of environmental lead parameters is not widely dispersed. The following is an example of a simple simulation using only default values.

From the main menu shown in Screen 2-1, enter "2" (Computation), then on the Computation Menu enter "1" (Run). The results shown on the monitor display the average of monthly geometric mean blood lead concentrations in one-year intervals, along with the daily lead uptakes from each medium in μg Pb/day. These results are the geometric mean blood lead concentrations and lead uptakes within each one-year age interval, assuming constant environmental lead concentrations from birth through each age interval. They can be interpreted as representing the results for a "typical" child in contact with these or similar lead concentrations. See Example 5-4 for an extension of this example to risk estimation.

5.2 APPLICATIONS WHERE ENVIRONMENTAL LEAD CONCENTRATIONS CHANGE OVER TIME

EXAMPLE 5-2. Reductions in Air and Dietary Lead Levels from 1975 to 1981 Decrease Baseline Blood Lead Concentrations

This example illustrates the estimation of historical exposures and baseline U.S. blood lead concentrations from 1975 to 1981.

- Air: The user should first enter the 1975 air lead levels from Figure 2-10.

- Diet: Then the user should enter the dietary lead values for the same time period, as in Table 2-1. However, no dietary lead intake values for children are shown for 1975 to 1977. We estimated the 0-11 month value for 1975 as 80 percent of the 1-year value for the 1978 value, that is 80 percent of 45.80 μg/d = 36.64 μg/d, since the 6-11 month dietary lead intake values for 1982-1984 are about 80 percent of the respective 1-year-old values. We then assumed that for a child born in 1975, the 1975 value was 36.64 μg/d, the 1976 value (age 1 year) was the same as the 1978 1-year-old value of 45.80 μg/d, the 1977 value (age 2 years) was the same as the 1978 2-year-old value of 52.90 μg/d, the 1978 value (age 3 years) was 52.70 μg/d as in Table 2-1, the 1979 value (age 4 years) was 47.30 μg/d as in Table 2-1, the 1980 value (age 5 years) was 38.70 μg/d as in Table 2-1. We assumed that the 1981 value (age 6 years) was 110 percent of the 1981 value at age 5 years or 110 percent of 35.80 μg/d = 39.38 μg/d, since the 1982-1984 6-year-old values are about 10 percent larger than the respective 5-year-old intake values. The input values for dietary lead intake are shown in Table 5-1.

- Water: Water lead concentrations were kept at the default values.

- Soil: Adjustments should be made for lead in soil and household dust. We assumed that soil lead levels, even in areas not heavily impacted by automobile traffic, would have been somewhat larger in 1975 than in 1981. In the absence of better information, we assumed that soil lead concentrations consist of two components, a genuine baseline of about 200 μg/g which is the current default, and a small increment from air lead deposition that adds about 100 μg/g soil lead per μg/m^3 air lead. This assumption implies a relatively small contribution of 10 μg/g to soil lead from current air lead levels of 0.1 μg/m^3. Thus the 1975 soil lead level is about 324 μg/g, the 1976 level about 322 μg/g, and so on, as shown in Table 5-2.

TABLE 5-1. USER-SELECTED ENTRIES FOR IEUBK MODEL WORKSHEET FOR EXAMPLE 5-2, CHILD BORN IN 1975

PARAMETER	DEFAULT VALUE	USER SELECTED OPTION	UNITS
DATA ENTRY FOR DIET (by year)			
Dietary lead intake			
Age =0-1 year (0-11 mo),	5.53	36.64	µg Pb /day
1-2 years (12-23 mo)	5.78	45.80	
2-3 years (24-35 mo)	6.49	52.90	
3-4 years (36-47 mo)	6.24	52.70	
4-5 years (48-59 mo)	6.01	47.30	
5-6 years (60-71 mo)	6.34	38.70	
6-7 years (72-84 mo)	7.00	39.38	

TABLE 5-2. USER-SELECTED ENTRIES FOR IEUBK MODEL WORKSHEET FOR EXAMPLE 5-2, CHILD BORN IN 1975

PARAMETER	DEFAULT VALUE	USER SELECTED OPTION	UNITS
DATA ENTRY FOR SOIL (by year)			
Soil lead concentration			
Age =0-1 year (0-11 mo) (1975)	0	324	µg/g
1-2 years (12-23 mo) (1976)	0	322	
2-3 years (24-35 mo) (1977)	0	320	
3-4 years (36-47 mo) (1978)	0	310	
4-5 years (48-59 mo) (1979)	0	290	
5-6 years (60-71 mo) (1980)	0	256	
6-7 years (72-84 mo) (1981)	0	247	

- Dust: The Multiple Source Analysis method for household dust should be used, since soil lead and air lead levels are changing over time. Since particles from leaded gasoline emission are believed to contribute significantly to surface soil transported into the house during these years, we have assumed that the soil-to-dust coefficient is 0.85 appropriate for this historical example, although the current default is 0.70, and the air-to-dust coefficient is 100. This was shown in Screen 2-10. These changes are reported to the user in the main Data Entry Screen for Soil/Dust.

The model can be run by returning to the Computation Menu and using Option 1, or by pressing the F5 key from any of the main media data entry screens. The results are shown on the display. The results are reasonably consistent with the decrease in child blood lead concentrations in the U.S. from about 15 μg/dL to 10 μg/dL found in the 1976-1980 NHANES II survey (U.S. Environmental Protection Agency, 1986). However, this exposure scenario follows a single child born in 1975 for six years through 1981. Direct comparison with NHANES II would require representative blood lead estimates for 1-year-olds in 1976, 2-year-olds in 1977, 3 year-olds in 1978 etc.

The importance of a worksheet in developing and documenting the exposure scenario should be clear to the reader. The worksheets for this example are shown in Tables 5-1 and 5-2. Since the exposure scenario here is for a typical urban child and is not specific to a site or neighborhood, the user should not try to extend these results for risk estimation purposes without incorporating interindividual variability and site-specific information concerning exposure variability.

The IEUBK model is a biokinetic model, and therefore has the ability to estimate changes in blood lead in response to yearly changes in environmental lead exposure for children of different ages. The following examples are presented to encourage the user to explore some of the IEUBK model's capabilities for evaluating age-dependent changes in lead exposure when this exposure changes over time.

EXAMPLE 5-3. Example for Children Moving From a Lower to a Higher Soil Lead Concentration

This example demonstrates the effects of change from a constant environmental lead concentration to a higher constant environmental lead concentration. Assume that a child

moved into a housing unit with a soil lead concentration of about 2000 μg/g, from a previous housing unit with a soil lead concentration of about 100 μg/g. Assume also that soil is a significant source of dust in household dust, and that the soil lead contribution to household dust lead is 70 percent of the soil lead concentration. The user can assess the maximum effect of new exposure to elevated soil lead (e.g., moving into a new residence). This assessment is for children of different ages, in an ordered sequence of runs. This sequence studies the effects of new exposure at later ages.

The work sheet for this example is similar to the segment shown in Table 5-2. In fact, a sequence of work sheets is needed to study the effects of moving at different ages. There are two variables to be considered here. The first variable is the age of the child, which is used in the IEUBK Model calculations, and is entered as the left-hand column of the work sheets. The second variable is the age at which the child moves into the new exposure environment. Thus, in Table 5-3(a), if the child moves at age 0 years, the child is exposed to 2000 μg/g lead in soil and 1400 μg/g lead in dust derived from soil from birth through age 6 years. However, if the child moves at age one year, the correct work sheet is shown in Table 5-3(b). In the work sheet in Table 5-3(b), the child is exposed to 100 μg/g lead in soil and 70 μg/g lead in household dust at age zero years, but to 2000 μg/g lead in soil and 1400 μg/g lead in dust from soil at ages 1 through 6 years. Similarly, if the child moves at age two years, the correct work sheet is shown in Table 5-3(c). In the work sheet in Table 5-3(c), the child is exposed to 100 μg/g lead in soil and 70 μg/g lead in household dust at ages 0 and 1 years, but to 2000 μg/g lead in soil and 1400 μg/g lead in dust from soil at ages 2 through 6 years.

The worksheets for Tables 5-3(a-c) are combined and shown as columns 2 to 4 in Table 5-3(d). The last 4 columns in Table 5-3(d) summarize the soil lead work sheet entries if the hypothetical child moves at ages 3, 4, 5, or 6 years respectively. For example, in the extreme right-hand column, if the child moves at age 6 years, he or she is exposed to 100 μg/g lead in soil from birth through age 5 years, then to 2000 μg/g at age 6 years.

The IEUBK Model simulation for this example is run 7 times, each run corresponding to a column in Table 5-3(d) or to a work sheet 5-3(a-c) or analogous work sheets for older children. The results, as annual averages of predicted geometric mean blood lead concentration, are shown in Table 5-4 in exactly the same order as in Table 5-3(d).

TABLE 5-3a. SOIL LEAD DATA ENTRY WORKSHEET FOR CHILD EXPOSED TO 2000 μg/g SINCE AGE 0 (BIRTH)

PARAMETER	DEFAULT VALUE	USER SELECTED OPTION	UNITS
DATA ENTRY FOR SOIL (by year)			
Soil lead concentration			
Age = 0-1 year (0-11 mo)	0	2000	μg/g
1-2 years (12-23 mo)	0	2000	
2-3 years (24-35 mo)	0	2000	
3-4 years (36-47 mo)	0	2000	
4-5 years (48-59 mo)	0	2000	
5-6 years (60-71 mo)	0	2000	
6-7 years (72-84 mo)	0	2000	

TABLE 5-3b. SOIL LEAD DATA ENTRY WORKSHEET FOR CHILD EXPOSED TO 2000 μg/g SINCE AGE 1

PARAMETER	DEFAULT VALUE	USER SELECTED OPTION	UNITS
DATA ENTRY FOR SOIL (by year)			
Soil lead concentration			
Age = 0-1 year (0-11 mo)	0	100	μg/g
1-2 years (12-23 mo)	0	2000	
2-3 years (24-35 mo)	0	2000	
3-4 years (36-47 mo)	0	2000	
4-5 years (48-59 mo)	0	2000	
5-6 years (60-71 mo)	0	2000	
6-7 years (72-84 mo)	0	2000	

TABLE 5-3c. SOIL LEAD DATA ENTRY WORKSHEET FOR CHILD EXPOSED TO 2000 μg/g SINCE AGE 2

PARAMETER	DEFAULT VALUE	USER SELECTED OPTION	UNITS
DATA ENTRY FOR SOIL (by year)			
Soil lead concentration			
Age = 0-1 year (0-11 mo)	0	100	μg/g
1-2 years (12-23 mo)	0	100	
2-3 years (24-35 mo)	0	2000	
3-4 years (36-47 mo)	0	2000	
4-5 years (48-59 mo)	0	2000	
5-6 years (60-71 mo)	0	2000	
6-7 years (72-84 mo)	0	2000	

TABLE 5-3d. WORKSHEET FOR YEARLY SOIL LEAD CONCENTRATION FOR HYPOTHETICAL CHILDREN MOVING FROM A RESIDENCE WHERE SOIL CONCENTRATION IS 100 μg/g TO A RESIDENCE WHERE SOIL CONCENTRATION IS 2000 μg/g

AGE OF CHILD (YEARS)	AGE AT TIME OF NEW EXPOSURE (YEARS)						
	0	1	2	3	4	5	6
0	2000	100	100	100	100	100	100
1	2000	2000	100	100	100	100	100
2	2000	2000	2000	100	100	100	100
3	2000	2000	2000	2000	100	100	100
4	2000	2000	2000	2000	2000	100	100
5	2000	2000	2000	2000	2000	2000	100
6	2000	2000	2000	2000	2000	2000	2000

TABLE 5-4. PREDICTED ANNUAL AVERAGE BLOOD LEAD CONCENTRATIONS (μg/dL) FOR HYPOTHETICAL CHILDREN MOVING FROM A RESIDENCE WHERE SOIL CONCENTRATION IS 100 μg/g TO A RESIDENCE WHERE SOIL CONCENTRATION IS 2000 μg/g

AGE OF CHILD (YEARS)	AGE AT TIME OF NEW EXPOSURE (YEARS)						
	0	1	2	3	4	5	6
0	16.2	2.8	2.8	2.8	2.8	2.8	2.8
1	18.6	16.3	3.0	3.0	3.0	3.0	3.0
2	17.7	17.7	14.5	2.8	2.8	2.8	2.8
3	17.3	17.3	17.2	13.5	2.6	2.6	2.6
4	14.7	14.7	14.7	14.5	10.2	2.3	2.3
5	12.6	12.6	12.6	12.6	12.2	8.6	2.1
6	11.3	11.3	11.3	11.3	11.2	10.8	7.5

The changes in exposure scenario are made by first using the parameter selection menu (Option "1" on the Main Menu), Option "4" on the parameter selection menu, and then entering selection "2" in the soil concentration box of the Soil/Dust menu. This allows the entry of separate values for soil lead exposure concentration at each age. The default value of 200 μg/g for each age may be replaced by 100 or by 2000, as indicated by the scenario on the work sheet. When finished, the user must return to the Soil/Dust menu. In order to change the dust lead exposure from the default, a constant 200 μg/g, the user must move the cursor down to the dust lead entry box in the Soil/Dust Menu and enter selection "3", the multiple source menu. The default soil-to-dust coefficient of 0.70 is activated by entering the Multiple Source Menu, and may be changed as needed. We will not modify either the soil-to-dust coefficient of 0.7, nor the air-to-dust coefficient of 100 μg/g per μg/m^3. The complete input file may be saved by returning to the Soil/Dust Menu and using the F6 key. The model may then be run by using the F5 key.

The results of the seven runs are shown in Table 5-4, which is analogous to Table 5-3(d). The second column shows blood lead concentrations for a typical child exposed to 2000 μg/g lead in soil since birth. The peak blood lead concentration of 18.6 μg/dL is reached at age one year. If the initial exposure to 2000 μg/g occurs later, the peak blood lead concentration is lower.

Most of the blood lead response to a change in exposure or a change in environmental lead concentration occurs in the first 3 months after the change. The change in blood lead during the first three months after changing exposure is at least 50 to 60 percent of the total difference in quasi-state-state blood lead concentration before and after the change. The remaining difference will slowly decrease during the next 2 years. We thus suggest that cross-sectional blood lead studies or baseline blood lead concentrations measured in longitudinal studies require that all children shall have lived at their present address for at least 3 to 6 months prior to the blood lead sample.

EXAMPLE 5-4. Example for Children in a Residence Where the Soil Has Been Abated

This sequence of runs considers soil lead exposure decreased from 2000 to 100 μg/g, and the soil contribution to dust decreased from 1400 to 70 μg/g, at ages 0 (i.e. constant exposure without soil and dust lead after birth), at age 1, age 2, and so on. This assessment studies the effects of abatement on children at different ages. The entries for this example are similar to those of Example 5-3. The summary of seven data entry worksheets is shown in Table 5-5(d), and the results are shown in Table 5-6.

TABLE 5-5a. SOIL LEAD DATA ENTRY WORKSHEET FOR CHILD WITH SOIL ABATED TO 100 μg/g SINCE AGE 0 (BIRTH)

PARAMETER	DEFAULT VALUE	USER SELECTED OPTION	UNITS
DATA ENTRY FOR SOIL (by year)			
Soil lead concentration			
Age = 0-1 year (0-11 mo)	0	100	μg/g
1-2 years (12-23 mo)	0	100	
2-3 years (24-35 mo)	0	100	
3-4 years (36-47 mo)	0	100	
4-5 years (48-59 mo)	0	100	
5-6 years (60-71 mo)	0	100	
6-7 years (72-84 mo)	0	100	

TABLE 5-5b. SOIL LEAD DATA ENTRY WORKSHEET
FOR CHILD WITH SOIL ABATED TO 100 μg/g SINCE AGE 1

PARAMETER	DEFAULT VALUE	USER SELECTED OPTION	UNITS
DATA ENTRY FOR SOIL (by year)			
Soil lead concentration			
Age = 0-1 year (0-11 mo)	0	2000	μg/g
1-2 years (12-23 mo)	0	100	
2-3 years (24-35 mo)	0	100	
3-4 years (36-47 mo)	0	100	
4-5 years (48-59 mo)	0	100	
5-6 years (60-71 mo)	0	100	
6-7 years (72-84 mo)	0	100	

TABLE 5-5c. SOIL LEAD DATA ENTRY WORKSHEET
FOR CHILD WITH SOIL ABATED TO 100 μg/g SINCE AGE 2

PARAMETER	DEFAULT VALUE	USER SELECTED OPTION	UNITS
DATA ENTRY FOR SOIL (by year)			
Soil lead concentration			
Age = 0-1 year (0-11 mo)	0	2000	μg/g
1-2 years (12-23 mo)	0	2000	
2-3 years (24-35 mo)	0	100	
3-4 years (36-47 mo)	0	100	
4-5 years (48-59 mo)	0	100	
5-6 years (60-71 mo)	0	100	
6-7 years (72-84 mo)	0	100	

TABLE 5-5d. WORKSHEET FOR HYPOTHETICAL CHILDREN IN A NEIGHBORHOOD WHERE SOIL CONCENTRATION IS REDUCED FROM 2000 μg/g TO 100 μg/g

AGE OF CHILD (YEARS)	AGE AT TIME OF ABATEMENT (YEARS)						
	0	1	2	3	4	5	6
0	100	2000	2000	2000	2000	2000	2000
1	100	100	2000	2000	2000	2000	2000
2	100	100	100	2000	2000	2000	2000
3	100	100	100	100	2000	2000	2000
4	100	100	100	100	100	2000	2000
5	100	100	100	100	100	100	2000
6	100	100	100	100	100	100	100

TABLE 5-6. PREDICTED BLOOD LEAD CONCENTRATIONS (μg/dL) FOR HYPOTHETICAL CHILDREN IN A NEIGHBORHOOD WHERE SOIL CONCENTRATION IS REDUCED FROM 2000 μg/g TO 100 μg/g

AGE OF CHILD (YEARS)	AGE AT TIME OF ABATEMENT (YEARS)						
	0	1	2	3	4	5	6
0	2.8	16.2	16.2	16.2	16.2	16.2	16.2
1	3.0	5.4	18.6	18.6	18.6	18.6	18.6
2	2.8	2.8	6.1	17.7	17.7	17.7	17.7
3	2.6	2.6	2.7	6.6	17.3	17.3	17.3
4	2.3	2.3	2.3	2.5	6.9	14.7	14.7
5	2.1	2.1	2.1	2.1	2.55	6.2	12.6
6	1.9	1.9	1.9	1.9	2.0	2.4	5.8

A sequence of work sheets is needed to study the effects of abatement at different ages. The two variables to be considered here are the child's age, which is a variable used in the IEUBK Model simulation, and the age of the child when the abatement was carried out, which is different for each run in the sequence of 7 runs. In Table 5-5(a), if the soil is abated at age 0 years, the child is exposed to 100 μg/g lead in soil and 70 μg/g lead in dust

derived from soil from birth through age 6 years. However, if the soil is abated at age one year, the correct work sheet is shown in Table 5-5(b). In the work sheet in Table 5-5(b), the child is exposed to 2000 μg/g lead in soil and 1400 μg/g lead in household dust at age zero years, but to 2000 μg/g lead in soil and 1400 μg/g lead in dust from soil at ages 1 through 6 years. Similarly, if the soil is abated at age two years, the correct work sheet is shown in Table 5-5(c). In the work sheet in Table 5-5(c), the child is exposed to 2000 μg/g lead in soil and 1400 μg/g lead in household dust at ages 0 and 1 years, but to 100 μg/g lead in soil and 70 μg/g lead in dust from soil at ages 2 through 6 years.

The worksheets for Tables 5-5(a-c) are combined and shown as columns 2 to 4 in Table 5-5(d). The last 4 columns in Table 5-5(d) summarize the soil lead work sheet entries for a hypothetical child if the soil is abated at ages 3, 4, 5, or 6 years respectively. For example, in the extreme right-hand column, if the soil is abated at age 6 years, he or she is exposed to 2000 μg/g lead in soil from birth through age 5 years, then to 100 μg/g at age 6 years.

The IEUBK Model simulation for this example is run 7 times, each run corresponding to a column in Table 5-5(d) or to a work sheet 5-5(a-c) or analogous work sheets for older children. The results, as annual averages of predicted geometric mean blood lead concentration, are shown in Table 5-6 in exactly the same order as in Table 5-5(d).

Abatement at age 1 reduces blood lead from 16.2 to 5.4 μg/dL in the first year after abatement, a reduction of 10.8 μg/dL or 66.7 percent. The effect at age 2 is a reduction from 18.6 to 6.1 μg/dL, that is 12.5 μg/dL or 67.7 percent. Abatement at age 5 has a reduction of 8.5 μg/dL or 57.8 percent in the first year. It should be noted that blood lead concentrations do not reach the post-abatement quasi-steady state level until two years after the abatement, so that the apparent reduction in blood lead concentration in the first year after abatement will underestimate the effectiveness of abatement.

EXAMPLE 5-5. Historical Exposure Reconstruction for Soil and Dust Lead Concentration and Dietary Lead Intake Around an Unused Lead Smelter

One of the issues that arose in developing validation case studies for the IEUBK model is that many of the earlier data sets were collected at sites where background lead exposure differed greatly from current default values, and where both background exposure and soil/dust exposure were changing substantially during the lifetime of the children in the blood

lead study. It was therefore necessary to construct an historical exposure scenario for the children in the blood lead study. The exposure reconstruction for the 1983 East Helena blood lead study was discussed in the initial validation of the UBK model (U.S. Environmental Protection Agency 1989). In this example, we will discuss the more complicated exposure situation for the 1983 companion study in the Silver Valley of Idaho. We rely heavily on the initial report on Kellogg Revisited (Panhandle District Health Department 1986), the Human Health Risk Assessment (Jacobs Engineering, 1989, for US EPA Region X), the Risk Assessment Data Evaluation Report (US EPA 1990), the House Dust Remediation Report (CH2M Hill 1991 for the Idaho Dept. of Health), the Record of Decision for the Bunker Hill site (U.S. Environmental Protection Agency 1991), and personal communications with Dr. Ian Von Lindern of Terragraphics Inc. (1992-1993).

The narrow east-west Silver Valley was divided initially into three residential areas, Area 1 (Smelterville) about 1.2 to 1.5 km northwest of the smelter complex, Area 2 (Kellogg) about 2.6 to 3.3 km east of the smelter complex, and Area 3 (Pinehurst) about 6 km west of the smelter complex. In subsequent studies this area was extended and subdivided into 5 to 11 areas or zones. A list of zones and distances is attached as Table 5-7. The main distinction is that the Page neighborhood which is only 3 km west of the smelter complex has been distinguished from Pinehurst, and that the Wardner neighborhood about 3 km southeast of the smelter complex has been separated from the Kellogg community. The five areas currently defined are closer in size and population to the "neighborhoods" recommended in Chapter 4.

Silver Valley has a complicated history of lead exposure, including significantly elevated air and dust lead exposures in 1974 and 1975, and a cessation of lead smelting activities after December 1991. Therefore, the exposure history reconstruction in Table 5-8 is a mixture of observed values and interpolated values. The observed values were sometimes recorded as geometric means and sometimes as arithmetic means, and as estimates or interpolations enclosed in brackets. The basis for the dust lead interpolation was not described in more detail in (Jacobs Engineering 1989). The soil lead concentrations were held at the last measured value until a new observed value had been achieved.

Soil and dust lead concentrations were only observed in 1974, 1975, 1983, and 1986-1988. Dust lead concentrations have also been observed in these communities since 1990. There are alternatives to estimating neighborhood soil and dust lead concentrations between actual observations, such as by linear interpolation, that may provide somewhat different estimates than the interpolations used in the human risk assessment study.

TABLE 5-7. NEIGHBORHOOD IDENTIFIERS AND DISTANCE FROM STACK FOR KELLOGG, ID, STUDY

ZONE	APPROXIMATE DISTANCE FROM ZONE CENTER TO Pb SMELTER STACK (Km)	DESCRIPTION
A	1.50	Smelterville, south of old Highway 110 and west of C street
B	1.15	Smelterville, east of C street
C	2.75	Kellogg, north of I-90 and west of Hill street
D	3.25	Kellogg, north of I-90 and east of Hill street
E	2.60	Kellogg, south of I-90 and west of Division street
F	3.30	Kellogg, south of I-90 and east of Division street
G	3.00	Wardner
H	5.70	Pinehurst
I	3.30	Page
J		Smelterville, (1974-75 only)
K		Kellogg/Page, (1974-75 (only)

An alternative assumption is that soil and dust lead concentrations decreased linearly between 1983 and 1986-1988. Thus, in Smelterville the decline in soil lead was 3047 - 2685 = 362 $\mu g/g$ in 4 years, or 90 $\mu g/g$ per year, whereas in Kellogg it was 2584 - 1988 = 596 $\mu g/g$ in 4 years, or about 150 $\mu g/g$ per year. The dust lead concentrations in Smelterville decreased by 3715 - 1203 = 2512 $\mu g/g$ in 5 years, or about 250 $\mu g/g$ per year, whereas the dust lead concentration in Kellogg decreased by 2366 - 1450 = 916 $\mu g/g$ in 5 years or about 230 $\mu g/g$ per year. However, the dust lead concentrations in 1990-1992 were still elevated above the Pinehurst concentration. It would be prudent to assume that the dust lead concentration was relatively constant for much of the period around and after 1986. By implication, since soil lead and air lead are sources for dust lead, one might assume that the soil lead and air lead concentrations for 1988-1992 are relatively constant at the 1988 values.

The soil and dust lead values for a Kellogg child born in 1983, assuming a linear decrease of 150 $\mu g/g$ in soil lead from 2584 $\mu g/g$ and a linear decrease of 230 $\mu g/g$ in dust lead from 2366 $\mu g/g$, is shown on the worksheet in Table 5-9. In this example we have

TABLE 5-8. OBSERVED AND ESTIMATED AIR, SOIL, AND DUST LEAD CONCENTRATIONS FOR USE IN HISTORICAL EXPOSURE RECONSTRUCTIONS IN SILVER VALLEY COMMUNITIES

YEAR	SMELTERVILLE			KELLOGG			PINEHURST		
	PbA[1,2]	PbS[1,2]	PbD[1,2]	PbA[1,2]	PbS[1,2]	PbD[1,2]	PbA	PbS	PbD
1971	5.7	[6141]	[3530]	8.2			[6.1]		
1972	11.2	[6141]	[6620]	9.6			[6.1]		
1973	16.5	[6141]	[12500]	15.0			[6.1]		
1974	14.3	6141	10583	14.0	2514	6581	6.1	765	2006
1975	8.9	3991	3533	7.4	2586	4573	3.1	508	1749
1976	9.8	[3991]	[6030]	7.5			3.4		
1977	9.1	[3991]	[5670]	6.8			3.6		
1978	5.4	[3991]	[3530]	5.4			2.7		
1979	6.6	[3991]	[4020]	5.9			3.1		
1980	6.2	[3991]	[3780]	5.9			2.2		
1981	4.6	[3991]	[2830]	4.1			1.2		
1982	0.88	[3991]	[3715]	0.28			0.16		
1983	0.20	3047	[3715	0.19	2584	2366	0.14	472	1155
1984	0.12	[3047]	[3715]	0.12			0.09		
1985	0.19	[3047]	[3715]	0.13			0.10		
1986	0.30	[3047]	[3715]	0.19			0.10		
1987	0.36	2685		0.17	1988		0.08		
1988	0.36	[2685]	1203[4]	0.11		1450[4]	0.08		
1989									
1990			1858[3]			1920[3]			1022[3]
1991			1496[3]			1502[3]			1068[3]
1992			978[3]			1227[3]			944[3]

Data Sources:

1. Jacobs Engineering (`9) for data before 1989. Tables 4-5, 4-7, 4-13. PbA values are arithmetic means of lead in air ($\mu g/m^3$), PbS and PbD values not in brackets are geometric means of lead in soil and dust ($\mu g/g$).
2. Jacobs Engineering (1989) for data before 1989. PbS and PbD values in brackets are estimates from Figure 4-16.
3. I. Von Lindern, personal communication. Arithmetic means of dust lead concentrations.
4. Record of Decision, 1991. Tables 5-1, 5-8.

TABLE 5-9. USER-SELECTED ENTRIES FOR IEUBK MODEL WORKSHEET FOR EXAMPLE 5-5, CHILD BORN IN KELLOGG, IDAHO, IN 1983

PARAMETER	YEAR	DEFAULT VALUE	USER SELECTED OPTION	UNITS
DATA ENTRY FOR SOIL (by year)				
Soil lead concentration				µg/g
Age = 0-1 year (0-11 mo)	1983	0	2,584	
1-2 years (12-23 mo)	1984	0	2,434	
2-3 years (24-35 mo)	1985	0	2,284	
3-4 years (36-47 mo)	1986	0	2,134	
4-5 years (48-59 mo)	1987	0	1,984	
5-6 years (60-71 mo)	1988	0	1,834	
6-7 years (72-84 mo)	1989	0	1,834	
DATA ENTRY FOR DUST (by year)				
Dust lead concentration				µg/g
Age = 0-1 year (0-11 mo)	1983	0	2,366	
1-2 years (12-23 mo)	1984	0	2,136	
2-3 years (24-35 mo)	1985	0	1,906	
3-4 years (36-47 mo)	1986	0	1,676	
4-5 years (48-59 mo)	1987	0	1,446	
5-6 years (60-71 mo)	1988	0	1,446	
6-7 years (72-84 mo)	1989	0	1,446	

treated soil and dust lead concentrations as typical values for the community. Model results for the distribution of blood lead concentrations using these inputs would be expected to be more narrow than seen in the actual community due to variability of exposure concentrations within the community.

The dietary lead intake depends on the age of the child and on the year of interest. For a child born in 1983, the dietary lead intake data entry worksheet is shown in Table 5-10, using data from Table 2-1. The two additional dietary exposure scenarios are for children who consume only home-grown vegetables, or only locally-caught fish. From Table 2-3 we calculate a weighted average lead concentration of 5.5 µg/g for leafy and root vegetables grown in Smelterville. The worksheet is shown in Table 5-11. From Table 2-4 we find a

TABLE 5-10. USER-SELECTED ENTRIES FOR IEUBK MODEL WORKSHEET FOR EXAMPLE 5-5, CHILD BORN IN SMELTERVILLE, IN KELLOGG, IDAHO, IN 1983

PARAMETER	DEFAULT VALUE	USER SELECTED OPTION	UNITS
DATA ENTRY FOR DIET (by year)			
Dietary lead intake			
Age = 0-1 year (0-11 mo),	5.53	14.42	µg Pb /day
1-2 years (12-23 mo)	5.78	22.67	
2-3 years (24-35 mo)	6.49	12.34	
3-4 years (36-47 mo)	6.24	9.08	
4-5 years (48-59 mo)	6.01	6.01	
5-6 years (60-71 mo)	6.34	6.34	
6-7 years (72-84 mo)	7.00	7.00	

TABLE 5-11. USER-SELECTED ENTRIES FOR IEUBK MODEL WORKSHEET FOR EXAMPLE 5-5

PARAMETER	DEFAULT VALUE	USER SELECTED OPTION	UNITS
DATA ENTRY FOR ALTERNATE DIET SOURCES (by food class)			
Concentration:			
home-grown fruits	0		µg Pb/g
home-grown vegetables	0	5.5	
fish from fishing	0	0.80	
game animals from hunting	0		
Percent of food class:			
home-grown fruits	0		%
home-grown vegetables	0	36	
fish from fishing	0	50	
game animals from hunting	0		

lead concentration in locally caught fish of 0.80 µg/g, over twice the national average level at that time. The data entry for fish is shown in Table 5-11. The assumed percentages for local vegetables and fish consumption are 36 and 5 percent, respectively.

The results for elevated soil and dust lead plus baseline dietary lead intake show that if locally-grown vegetables and fish are consumed in large amounts, there is a modest increase in blood lead concentration at each age.

We will discuss blood lead estimation for this example in the validation studies that will reported separately from this manual. We have included this example in the Guidance Manual to give the reader some "real world" exposure scenarios and to confront the reader with some of the choices that may need to be made in developing historical exposure scenarios for blood lead studies.

5.3 APPLICATIONS FOR PROBABILITY AND RISK ESTIMATION

EXAMPLE 5-6. Default Parameters

For the default parameters in Example 5-1, the estimated geometric mean blood lead for children of ages 24 to 35 months is 4.2 μg/dL. The user may choose any other age range. If the user next goes into Option 1 from the bottom menu, then "3" from Graphics Selection Menu and selects age range 24-36 months (K), the log-normal probability density should appear on screen. This plot can be printed on a user-specified printer. The user can save the graphics file for additional review using the Multiple Runs Option M with just a single run. No default parameters were changed, except for the GSD, which was changed to 1.42. With GSD = 1.42, there is an estimated 0.68 percent risk that a child with the default exposure scenario will have a blood lead exceeding 10 μg/dL.

A useful alternative display is shown by selecting the Distribution Probability Percent "2" among the plot options. This shows the risk of a blood lead exceedance for any possible blood lead concentration from 0 to 16 μg/dL, not just the level of concern of 10 μg/dL, but the line is too close to zero to be visually distinctive above 12 μg/dL.

EXAMPLE 5-7. Sensitivity of Risk Estimates to User-Selected Geometric Standard Deviation

One way to carry out sensitivity analyses is to carry out each simulation run individually, but to collect the results for different parameters in cumulative output data sets.

The IEUBK model does not currently offer options to do this for any parameters except media concentrations that do not change with age during single simulation run. We will thus fix all of the model parameters at default values, except for the GSD, which in this example will take values from 1.42 to 1.90. After running the model as in the preceding example, we will select "6" in the Graphics Selection Menu. This allows the user to change both the GSD and the blood lead level of concern, while leaving the geometric mean blood lead level at the same value, here 4.2 μg/dL. The results for different GSD values are shown in Table 5-12, for children of ages 24-35 mos.

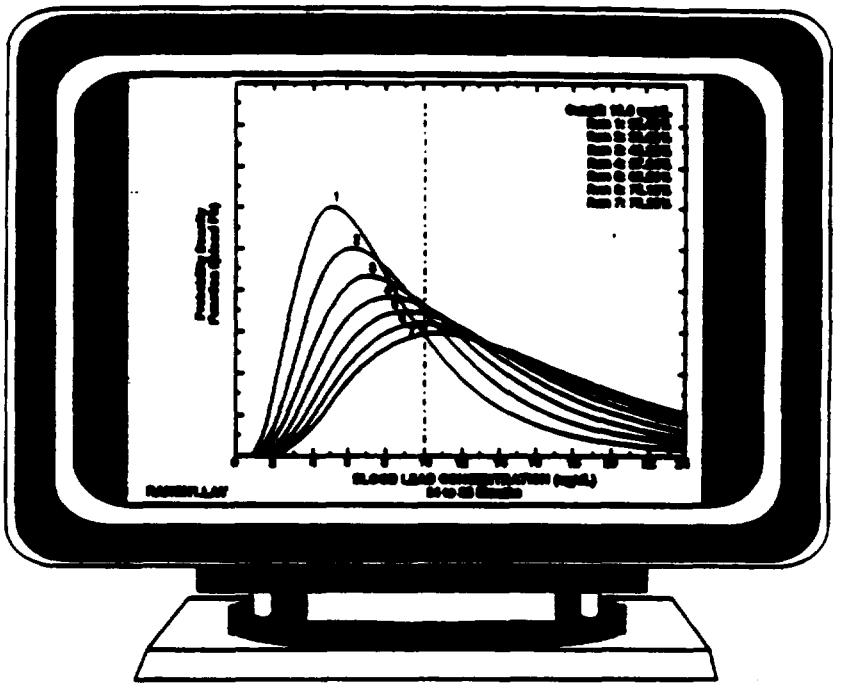
TABLE 5-12. EFFECTS OF GSD ON THE PROBABILITY OF EXCEEDING 10 μg/dL, USING ONLY DEFAULT EXPOSURE PARAMETERS, FOR CHILDREN AGES 24 TO 35 MONTHS

GSD	Probability of Blood Lead > 10 μg/dL
1.42	0.0068
1.50	0.0157
1.60	0.0324
1.70	0.0513
1.80	0.0696
1.90	0.0870

EXAMPLE 5-8. Effects of Dust Lead Concentration on Risk Estimates for Fixed Soil Lead Concentration

In this example, we can use Option "2" on the Computation Menu to assess the effects of different dust lead levels for a fixed soil lead concentration. We will here assume a soil lead concentration of 1,000 μg/g, and dust lead concentrations incremented in the Multiple Runs Analysis. The soil lead concentration is not a default and must be reset to 1,000 μg/g in the Soil/Dust Data Entry Menu (4). We will use 7 levels of dust lead, from 0 to 1,500 μg/g by steps of 250 μg/g. These should be changed in the Multiple Runs Analysis, by entering sub-menus 1 (medium = dust), 2 (range set to 0-1500), and 4 (7 levels of dust, results sent to graphics and results save files). All of the other parameters are set to default values except for a GSD of 1.70 to illustrate the effect of a larger GSD. Selection 3 runs the models.

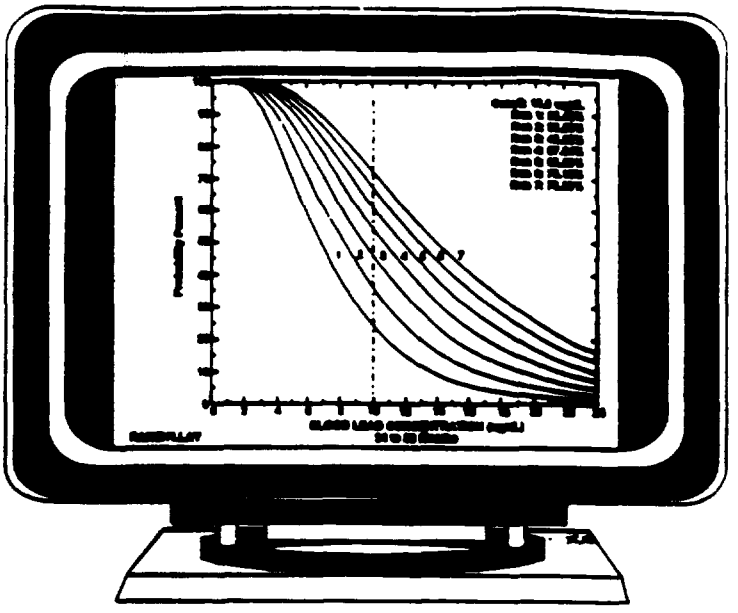
Return to the Output Menu (3), select Plot (2), select Plot Overlay (Density), highlight overlay file, select 24-36 months (K), and the plot will appear on the display. The results are shown in Screen 5-1, which shows the probability density plots for a GSD of 1.70. We are assuming maximum bioavailability (30%). With no lead in dust, the probability that a 2-year-old will exceed 10 μg/dL is estimated as 25 percent. (Run 1), whereas with dust lead concentration of 1,500 μg/g (1.5 times as large as the soil lead concentration) this probability increases to 73 percent. We see that there is substantial sensitivity to the soil-to-dust coefficient and to additional non-soil sources of dust lead in this example.



Screen 5-1. Multiple runs probability density function for soil lead = 1,000 μg/g, dust lead = 0 to 1,500 μg/g, by steps of 250 μg/g (Runs 1 through 7) in Example 5-6.

The cumulative exceedance probability plots (selection 4 in the Graphics Selection Menu) are shown in Screen 5-2. These plots show a clear increase of risk with increasing dust lead level at all blood lead levels of concern, and offer the user a visual display that may help to separate the risk estimates for different dust lead levels.

In order to assess the relationship between geometric mean blood lead and dust lead concentration, the user must set soil lead to 1000 in Option 4 of the Parameter Input Menu

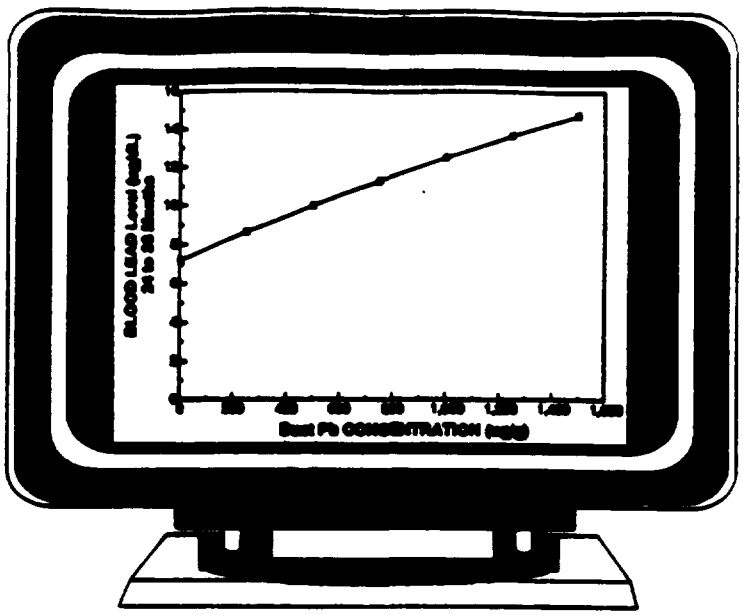


Screen 5-2. Multiple runs probability of exceedance of blood lead levels for soil lead = 1,000 μg/g, dust lead = 0 to 1,500 μg/g, by steps of 250 μg/g (Runs 1 through 7) in Example 5-6.

and then go to Option "2" of the Computation Menu. In Option B, enter sub-menus 1 (medium = dust), 2 (range set to 0-1500), and 4 (7 levels of dust, results sent to graphics and results save files). All of the other parameters are set to default values. Selection 3 runs the models. The results may be plotted immediately, as shown in Screen 5-3, or saved in a *.PBM file for later plotting. Note the slight nonlinearity as dust lead levels exceed 1,000 μg/g, due to saturable absorption effects.

5.4 BATCH MODE INPUT AND STATISTICAL ANALYSES OF OUTPUT

This section demonstrates the use of the batch mode analysis method with input data that are typical of the data available to the user in most environmental lead field studies. Assessment of goodness of fit of predicted and observed blood lead levels (when available) requires a statistical analysis of the data using a variety of mathematical and graphical techniques. Output data from the batch mode runs are in ASCII files that can be loaded into almost any statistical analysis package or spreadsheet program that the user may want to use.



Screen 5-3. Relationship of predicted blood lead to dust lead in Example 5-6.

The IEUBK batch mode output files will require little or no editing before being imported into other programs, unless the missing value code (---) is incompatible with the user's package. We have provided a small special-purpose program called PBSTAT that can be used after the batch mode output file is created, by exiting from the IEUBK model and executing PBSTAT, or by Option "5" in the Batch Mode Menu. PBSTAT is provided as a convenience for the user who may not have or wish to use other programs with the IEUBK output file. The statistical and graphical methods in PBSTAT are demonstrated in the following examples. Additional statistical analyses of the batch mode output data files are not possible using PBSTAT, and must be done with other programs.

EXAMPLE 5-9. Complete Data Set for an Old Mining Community

The input data format for a batch mode input file was described in Section 3.3. The data input file for this example is shown in Table 3-2. This data set was produced by a computer simulation and was edited into the format shown in Table 3-2. These are complete data, i.e., there are no missing values for any of the variables.

Let us suppose that these data represent the data for a sample cohort of children, all of whom were 18 months old at the time of blood lead sampling in late October. Let us assume that the data were collected in the community of "Mountain Pass", an old historic town that has been the site of active lead mining, ore processing, and smelting operations for over 100 years. These operations stopped about 25 years ago, and after a period of declining population the town is once more growing as the center of newly developed tourist and outdoor recreation activities. There is now considerable concern about the potential risk of elevated lead concentrations in soil and in the interior dust of the older houses in Mountain Pass. These children were recruited in the first phase of a long-term prospective study on changes in blood lead concentration in Mountain Pass children during a proposed soil lead abatement project.

The data set contains blood lead concentration in children, soil and dust lead concentration in their houses, in four neighborhoods in Mountain Pass. Air lead concentration were measured by a Total Suspended Particulate (TSP) sampler about ten years ago and were found to be less than 0.2 $\mu g/m^3$, so have not been measured since then. First-draw and partially flushed water lead samples were collected at each house, but have not yet been analyzed. Lead-based paint was measured by a portable X-Ray Fluorescence Spectrophotometer (XRF), but there have been some concerns about the instrument calibration during the unseasonably cold weather in which the measurements were made and the site manager has decided not to use the XRF data until the XRF measurements can be replicated next summer. (Even though this is only a hypothetical example, the reasons why some data may not be available are real, and are all too likely to occur in any real field study). The first model run done by the site manager used this data set "as is", with all of the parameters set to their default values in Table 3-1.

The batch mode run is made from Option 4 in the Computation Menu. The user must identify the input data set, known here as EXAMPLE1.DAT. The user also has the option of renaming the data set before running the batch mode analysis. If the user does not rename the data set, then [name].DAT input file results will be saved in data sets [name].TXT and [name].ASC—in this case, EXAMPLE1.TXT and EXAMPLE1.ASC. The output data file EXAMPLE1.TXT may be viewed from Option "2" of the Batch Mode Menu after the batch run is completed.

Option "5" of the Batch Mode Menu, can be used to examine the differences between observed and predicted blood lead levels using a variety of graphical and statistical techniques. The user must leave the main IEUBK model in order to enter the statistical and

graphical program PBSTAT. Selection 1 in the PBSTAT menu allows the user to load the ASCII file denoted [name].ASC. Selection 2 displays a screen full of statistical information. The information on this screen should be useful for many reports. The table includes the geometric and arithmetic mean blood lead concentrations, as well as the 25th, 50th (median), 75th, and 90th percentiles of observed and predicted blood lead levels. This screen reports paired-sample T-tests for the equality of geometric mean observed and predicted blood lead levels in the neighborhood, which is a test of the equality of the mean logarithms (left side of screen). Tests of the equality of the arithmetic mean blood lead concentration are shown on the right-hand side. You should not expect that the statistical tests will report agreement between observed and predicted values (see Section 1.1.5.3). These tests are used to help diagnose problems.

The two-sample Kolmogorov-Smirnov (denoted K-S) test of the equality of the two distribution functions is also reported. This is based on a very simple statistic, the largest absolute difference between the cumulative distribution of the observed blood lead levels and the cumulative distribution function of the predicted values. We have knowingly violated the assumption that these values are independent, thus the null hypothesis distribution will not give valid significance levels. However, we have found that the K-S statistic, together with the percentiles, provides valuable information about the kinds of discrepancies between the neighborhood-scale blood lead distribution and the distribution of predicted blood lead concentration.

Graphical comparisons of observed and predicted blood lead concentrations are very helpful. If the user exits from the statistics screen and then uses Selection 3 in the PBSTAT selection menu, for graphics and plots, there are a number of choices. Option 1 in the PBSTAT graphics selection menu allows plots of cumulative distribution functions, either singly or combined. Either regular or log-transformed blood lead concentrations may be plotted. The empirical cumulative distribution functions (CDF's) differ substantially. Another useful graphical comparison is in Selection 4 of the Graphing Selection menu, "box and whisker" plots. The boxes show the quartiles of the distribution(s), and the whiskers show the range of non-outlier blood lead concentrations. Outliers, by internal criteria, are shown as isolated data points. Observed and predicted values are highly correlated in the example, as shown by Graphing Selection choice 2. Many other plots may be generated by use of Selection 3.

In this example the model has somewhat over-estimated the observed blood lead concentrations. Any one of several factors could explain the difference between observed

and predicted blood lead concentrations in these children. Are there adequate quality assurance data for both the blood lead and the environmental lead measurements and do they show satisfactory performance during the study? Because the narrative for this scenario stated that blood lead concentrations were collected in late October, which was described as "unseasonably cold", could the children have been spending much less time playing in soils outside? If so, the blood lead data may reflect lower-than-average intake of soil recently, so that the ingestion rates in the model, which are annual averages, are not representative of the atypical conditions under which these blood lead data were collected. Were most of these children placed in some sort of day-care facility? If so, then the children in the day-care facilities could be analyzed as a separate group with appropriate lead concentration data for the facilities. Other possibilities, such as lower bioavailability of soil lead at some houses or in some neighborhoods, should be investigated. In any event, the answers to these questions are going to be found in site-specific data about child behavior, exposure to soil and dust, and on the chemical and physical properties of the soil and dust at the site, and not in further manipulations of model parameters. An analysis of these data, with additional exposure data, is presented as Example 5-11.

EXAMPLE 5-10. Batch Input Data File with Missing Environmental Lead Data

Some environmental data in a data set may be missing because the samples were not collected, were lost or damaged during transportation, storage, and sample preparation for analysis, or were improperly coded and thus not recorded. In any case, the values for missing data in an IEUBK model batch mode input file may be coded by an isolated decimal point where the variable value would otherwise be placed. Examples are given in the data sets EXAMPLE2.DAT and EXAMPLE3.DAT provided on the program disk. Missing values for water lead, air lead, and paint lead are automatically replaced by default values: 4 μg/L for water, 0.1 $\mu g/m^3$ for air, and 0 μg/day for alternative sources. The imputation method for soil and dust lead is different. If soil lead is missing, and dust lead is not missing, then the missing value of soil lead is set to the dust lead value. If dust lead is missing, and soil lead is not missing, then the missing value of dust lead is set to the soil lead value. These cases may be used to estimate or predict blood lead levels. If both soil and dust lead concentrations are missing, then no data are imputed and the blood lead concentration is not calculated for this child. The missing values imputed by the model are earmarked by an asterisk in the [name].TXT output file. The user is responsible for defining an appropriate data imputation process for any site-specific data set that has missing values. The file along with any imputed data should be created before it is submitted to the Batch Mode Option.

One convenient method for imputation of missing dust lead levels is to invoke the Multiple Source menu alternative for dust. The default values in this option (soil-to-dust coefficient of 0.70, air lead contribution of 10 μg/g to house dust) produce a somewhat different set of dust lead estimates and correspondingly different predicted blood lead concentrations.

Note that missing values of blood lead do not affect the prediction of blood lead from environmental lead data, provided that either a soil lead or a dust lead concentration is present, or that the user has imputed values for soil and dust lead calculated by some other method and inserted in place of the missing value.

EXAMPLE 5-11. Lead Exposure in an Old Mining Community Using Site-Specific Information About Ingestion of Soil and Dust

Suppose that the site manager in Example 5-9 has obtained additional information about the children in this sample, and finds that almost all of them have been enrolled in a day care program in this community. Upon visiting the day care facility, the site manager observes that the facility is modern, with easily cleanable floors, entrance surfaces and window sills. She or he observes that the facility appears to be cleaned often, and that the day care facility operators are aware of the hazard of childhood exposure to lead in dust and are making deliberate efforts to reduce the exposure. She or he also learns that most of the children's parents are employed full-time, and that most of these children spend 8 to 10 hours per day at the facility.

Is there now enough information to change the parameters of the IEUBK model so as to possibly provide a closer description of the data? We would not recommend rerunning the IEUBK Model without additional site-specific data. If predicted blood lead concentrations tend to be somewhat larger than those observed, any one or more of the following possibilities could explain the discrepancy:

(i) The soil lead and dust lead concentrations at the day care center may be much lower than the residential lead concentrations, so that a significant part of the child's daily ingestion of soil and dust includes much less lead than if the same quantity were ingested at home;

(ii) The quantity of soil and dust ingested may be smaller than expected because the child spends a great deal of time away from the home in a relatively clean environment, and frequently interacts with adult

caretakers and with other children, thereby reducing both environmental and behavioral magnifiers of soil and dust ingestion;

(iii) The bioavailability of lead in soil and dust at home or elsewhere may be lower than the default values used in the IEUBK Model;

(iv) The children in the sample may represent a non-typical sub-population with respect to ingestion or absorption;

(v) There may be measurement errors in soil lead, dust lead, or blood lead, possibly causing a systematic downward bias in lead measurements.

Any manipulation of the IEUBK Model that reduces lead uptake from a medium would reduce the predicted blood lead concentration and improve the overall fit of the predicted values to the observed values. This does not prove that the manipulation is valid. Lead uptake is the product of ingestion rate and absorption from the medium, so that achieving goodness of fit to the observed values can never prove the correctness of the manipulation of parameters.

We would recommend that some additional site studies be carried out to evaluate these possible causes. These studies include, in the same sequence (i-v):

(i) The soil lead, dust lead, and drinking water lead concentrations at the day care center should be measured;

(ii) The amount of dust in both the residence units and the day care center should be determined by measuring floor dust loadings;

(iii) Methods for child recruitment should be evaluated for possible sampling biases. Socio-demographic factors that may affect soil and dust ingestion should be investigated, including the role of parental awareness and public information programs. Nutritional differences that may affect lead bioavailibility, such as deficiency or repleteness of calcium intake, should be determined where feasible;

(iv) Seasonal biases, biases in sampling locations and in timing of soil and dust sampling studies should be considered as possible measurement errors. QA/QC data for analytical procedures for soil lead, dust lead, blood lead and other media should be reviewed for possible errors, instrument drift or other systematic biases.

For risk assessment applications, it may be preferable to use the default exposure scenario for children who do not spend most of their waking day in a clean environment outside the home. There is no guarantee that other children in this community will not be at

higher risk than the children in the sample. We are not suggesting the use of conservative assumptions about ingestion, but rather, the use of realistic assumptions about a plausible alternative exposure scenario (for example, if the day care facility closes down and is not replaced by a similar facility).

5.5 SOIL LEAD ABATEMENT EXAMPLES

Example 5-12. Use of the Multiple Runs Selection to Estimate Soil Lead Abatement Target Levels when Household Dust is Also Allowed to Vary

One of the more frequent applications of the IEUBK model has been to help determine soil lead concentrations for which abatement may be needed in order to reduce the likelihood of exceeding a blood lead level of concern (LOC) to some user-defined risk of exceedance (ROE) of the LOC at the site. These soil lead target concentrations are site-specific variables and reflect to a greater or lesser degree all of the other parameters that determine childhood blood lead levels after abatement. Effective soil lead abatement will often include household dust abatement, both to remove historical reservoirs of contaminated household dust and to help maintain lower household dust lead concentrations after soil abatement. In this situation, the post-abatement environment must be characterized by a site-specific soil-to-dust coefficient so that the soil lead target concentration is connected to a post-abatement dust lead concentration using the Multiple Source Analysis in the Soil/Dust Data Entry Menu. In this example, we will assume that all of the parameters in the model have been set to default values, but even if the default selections in the Multiple Source Analysis for household dust are invoked, they will not be activated without selecting the Multiple Source option. The following steps are used to illustrate soil target levels for a soil-to-dust coefficient of 0.70 and an air-to-dust coefficient of 100 μg Pb/g dust per μg Pb/m^3 air.

1. From the Main Menu, use Option 1: Parameter Menu, then Option 4: Soil/Dust Data Entry Menu, then tab down to Line 2 (Indoor Dust Pb) and use Option 3: . ultiple Source Analysis.

2. The user may select the soil-to-dust coefficient other than 0.70 and the air-to-dust coefficient other than 100, but even if the default values are used the user must enter this menu and then Escape back to the Soil/Dust Entry Menu.

3. Escape (exit) from the Soil/Dust Data Entry Menu to the Parameter Menu, then to the Main Menu. Choose Option 2: Computation Menu, then Option 2: Multiple Runs. This will put the user into the RANGE SELECTION MENU.

4. Set up a range-finding run by using Options 1, 2, and 4 in the Range Selection Menu. In Option 1 (Media), choose Soil and return to the Range Selection Menu. In Option 2 (Range), choose Start = 0 (0 μg/g soil lead) and End = 1500 (1500 μg/g soil lead) and return to the Range Selection Menu. In Option 4 (Output Choices), respond "Yes" to the query "Send to Overlay File", respond "7" to the query "Number of Runs for Range". This will produce output runs at 7 equally spaced levels of soil lead from 0 to 1500 μg/g, namely at 0, 250, 500, 750, 1000, 1250, and 1500 μg/g. The user who is not familiar with this option may also wish to respond "Yes" to the query "Display summary outputs". Return to the Range Selection Menu.

5. Run the Multiple Runs Analysis by selecting Option 3 on the Range Selection Menu. The user should see the message that the data sets RANGE#.LAY and RANGE#.TXT have been saved. The data set RANGE#.LAY is needed to obtain the probability plot values. The data set RANGE#.TXT is needed to document the input parameters for the run.

6. In order evaluate the range-finding runs, exit from the Range Selection Menu to the Computation Menu, then to the Main Menu. Select Option 3: Output Menu, then Option 2: Plot menu, then select the GSD and the blood lead level of concern (LOC). The default values GSD = 1.60 and LOC = 10 μg/dl are used here, so no selection is necessary; otherwise, use Option 6. Then use Option 5: Plot Overlay File (probability density functions). Tab down and select the appropriate RANGE#.LAY file, then select the age range "H", ages 0-84 months, or any other range, as needed. The probability of exceeding blood lead 10 μg/dL for each soil lead concentration from 0 to 1500 μg/g by steps of 250 μg/g is shown in Table 5-13.

TABLE 5-13. RANGE FINDING RUN FOR TARGET SOIL LEAD CONCENTRATION

OVERLAY PLOT	SOIL LEAD CONCENTRATION (μg/g)	PROBABILITY OF EXCEEDING 10 μg/dL, percent
1	0	0.00
2	250	1.99
3	500	12.03
4	750	26.86
5	1000	42.68
6	1250	55.50
7	1500	64.01

7. As a result of the range-finding runs shown in Table 5-13, the soil lead target concentration is between 250 μg/g (ROE = 1.99 %) and 500 μg/g(ROE = 12.03%). In order to narrow the list of possible values, repeat steps 4, 5, and 6 with a smaller range of values. We selected Start = 320 μg/g and End = 420 μg/g in Option 2 (Range) of the Range Selection Menu, and selected 6 runs in Option 4 of the Range Selection menu. Run the Multiple Runs Analysis with Option 3. This produces an output data set RANGE#+1.LAY. Plot the results in RANGE#+1.LAY for soil lead concentrations of 320, 340, 360, 380, 400, and 420 μg/g. The results are shown in Table 5-14.

TABLE 5-14. FOCUSED RUN FOR TARGET SOIL LEAD CONCENTRATION

OVERLAY PLOT	SOIL LEAD CONCENTRATION (μg/g)	PROBABILITY OF EXCEEDING 10 μg/dL, percent
1	320	3.24
2	340	3.45
3	360	3.90
4	380	4.15
5	400	4.70
6	420	5.00

8. Table 5-14 shows that the highest value of 420 μg/g appears to produce ROE = 5.00%. To confirm this, repeat Step 7 with a much smaller range of values. We selected Start = 400 μg/g and End = 430 μg/g in Option 2 (Range) of the Range Selection Menu, and selected 4 runs in Option 4 of the Range Selection menu. Run the Multiple Runs Analysis with Option 3. This produces an output data set RANGE#+2.LAY. Plot the results in RANGE#+2.LAY for soil lead concentrations of 400, 410, 420, and 430 μg/g. The results are shown in Table 5-15. **This procedure has identified a soil lead concentration of 410 μg/g as the target level.**

TABLE 5-15. VERIFICATION RUN FOR TARGET SOIL LEAD CONCENTRATION

OVERLAY PLOT	SOIL LEAD CONCENTRATION (μg/g)	PROBABILITY OF EXCEEDING 10 μg/dL, percent	DUST LEAD CONCENTRATION (μg/g)
1	400	4.70	290
2	410	5.00	297
3	420	5.00	304
4	430	5.32	311

9. The user may wish to view the dust lead concentrations corresponding to this procedure. In order to view RANGE#+2.TXT, return to the Main Menu, then the Computation Menu and select Option 4: Batch Mode. Select Batch Mode Option 2: View TXT File, the RANGE#+2.TXT. The dust lead concentrations are shown in the last column of Table 5-15.

6. REFERENCES

Agency for Toxic Substances and Disease Registry (1988). Nature and extent of childhood lead poisoning in the United States: A Report to Congress. U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA.

Alexander, F. W.; Clayton, B. E.; Delves, H. T. (1974). Mineral and trace-metal balances in children receiving normal and synthetic diets. Q.J. Med. 43: 89.

Alexander, F. W. (1974). The uptake of lead by children in differing environments. Environ. Health Perspectives 73: 155-159.

Alexander, F. W.; Delves, H. T.; Clayton, B. E. (1973). The uptake and excretion by children of lead and other contaminants. In: Barth, D.; Berlin, A.; Engel, R.; Recht, P.; Smeets, J. eds. Environmental health aspects of lead: proceedings, international symposium; October 1972; Amsterdam, The Netherlands. Luxembourg: Commission of the European Communities; pp. 319-331.

Allott, R. W.; Hewitt, C. N.; Kelly, M. R. (1990). The environmental half-lives and mean residence times of contaminants in dust for an urban environment: Barrow-in-Furness. Sci. Total Environ. 93: 403-410.

Altman, P. L.; Dittmer, D. S. (eds) (1972). Biological Data Book, 2nd Ed. pp 195-201. Bethesda, MD: Federation of American Societies for Experimental Biology.

American Academy of Pediatrics (1972). Lead content of paint applied to surfaces accessible to young children. Pediatrics 49(6): 918-921.

American Water Works Association Research Foundation (AWWARF) (1988). Review of the Biological Basis of the Proposed Drinking Water Lead Standard. Prepared by Karch and Assoc., Inc. Washington DC.

Amitai, Y.; Brown, M. J.; Graef, J. W.; Cosgrove E. (1991). Residential deleading: Effects on the blood lead levels of lead-poisoned children. Pediatrics 88: 893-897.

Aungst, B. J.; Fung, H. (1981). Kinetic characterization of in vitro lead transport across the rat small intestine. Toxicol., Appl. Pharmacol. 61: 39-57.

Baes, C. F.; Sharp, R. D.; Sjoreen, A. L.; Shor, R. W. (1984). A Review of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture. Report prepared by Oak Ridge National Laboratory (Martin Marietta) for U.S. Dept. of Energy under U.S. EPA Contract #EOA-78-D-X0394 (AD-89-F-2-A106).

Bander, L. K.; Morgan, K. J.; Zabik, M. E. (1983). Dietary lead intake of preschool children. Am. J. Public Health 73(7): 789-794.

Barltrop, D.; Meek, F. (1979). Effect of particle size on lead absorption from the gut. Arch. Environ. Health July/Aug. 1979.

Barltrop, D.; Khoo, H. E. (1975). The influence of nutritional factors on lead absorption. Postgrad Med. J. 51: 795-800.

Barltrop, D.; Meek, F. (1975). Absorption of different lead compounds. Postgrad. Med. J. 51: 805-809.

Barltrop, D.; Thornton, I.; Strehlow, C. D.; Webb, J. S. (1975). Absorption of lead from dust and soil. Postgrad Med. J. 51: 801-804.

Barry, P. S. I. (1981). Concentrations of lead in the tissues of children. Br. J. Ind. Med 38: 61-71.

Barton, J. C., Conrad, M. E.; Harrison, L.; Nuby, S. (1978). Effects of iron on the absorption and retention of lead. J. Lab. Clin. Med. 92: 536-547.

Barton, J. C.; Conrad, M. E.; Harrison, L.; Nuby, S. (1980). Effects of vitamin D on the absorption and retention of lead. Am. J. Physiol. 238; G124-G130.

Bentler, P. M., *EQS Structural Equations Program Manual*. BMDP Statistical Software, Los Angeles CA. 1989.

Binder, S.; Sokal, D.; Maughan, D. (1986). Estimating soil ingestion: The use of tracer elements in estimating the amount of soil ingested by young children. Arch. Environ. Health 41: 341-345.

Blake, K. C. H.; Barbezat, G. O.; Mann, M (1983a). Effect of dietary constituents on the gastrointestinal absorption of ^{203}Pb in man. Environ. Res. 30: 182-187.

Blake, K. C. H.; Mann, M. (1983b). Effect of calcium and phosphorus on the gastrointestinal absorption of ^{203}Pb in man. Environ. Res. 30:188-194.

Bollen, K. A. (1990). Structural equations with latent variables. New York: John Wiley and Sons.

Bornschein, R. L.; Clark, C. S.; Pan, U. W.; Succop, P. A. et al. (1990). Midvale Community Lead Study. Department of Environ. Health, University Cincinnati Medical Center. July 1990.

Bornschein, R. L.; Clark, C. S.; Grote, J.; Peace, B.; Roda, S.; Succop, P. A. (1988). Soil lead—Blood lead relationship in a former lead mining town. In: Environmental Geochemistry and Health, Monograph Series 4, Lead in Soil: Issues and Guidelines. (Eds) B. E. Davies and B. G. Wixson. Science Review Limited, Northwood, England. pp. 149-160.

Bornschein, R. L.; Succop, P. A.; Krafft, K. M. et al. (1986). Exterior surface dust lead, interior house dust lead and childhood lead exposure in an urban environment. In: Trace Substances in Environmental Health II—A Symposium. (Ed) D. D. Hemphill, University of Missouri, Columbia MO. pp. 322-332.

Bornschein, R. L.; Succop, P.; Dietrich, R. N.; Clark, C. S.; Que Hee, S.; Hammond, P. B. (1985). The influence of social and environmental factors on dust lead, hand lead, and blood lead levels in young children. Environ. Res. 38: 108-118.

Bruenger F. W.; Stevens, W.; Stover, B. J. (1973). The association of ^{210}Pb with constituents of erythrocytes. Health Physics 25: 37-42.

Brunekreef, B. (1984). The relationship between air lead and blood lead in children: A critical review. Sci. Total Environ. 38: 79-123.

Brunekreef, B.; Dook, N.; Clausing, P. (1987). Variability of exposure measurements in environmental epidemiology. Amer. J. Epidem. 125: 892-898.

Buncher, C. R.; Succop, P. A.; Dietrich, K. N. (1991). Structural equation modeling in environmental risk assessment. Environ. Health Persp. 90: 209-213.

Bushnell, P. J.; DeLuca, H. F. (1983). The effects of lactose on the absorption and retention of dietary lead. J. Nutr. 113: 365-378.

Calabrese, E. and Stanek, E. J. (1991). A guide to interpreting soil ingestion studies. II. Qualitative and quantitive evidence of soil ingestion. Reg Toxicol Pharmacol 13: 278-292.

Calabrese, E.; Stanek, E. J.; Gilbert, C. E. (1991). Evidence of soil-pica behavior and quantification of soil ingested. Human Experi. Toxicol. 10: 245-249.

Calabrese, E. Qualitative and quantitative evidence of soil ingestion. Presented at the Symposium on the Bioavailability and Dietary Uptake of Lead. Sept. 24-27, 1990. Chapel Hill, NC.

Calabrese, E.; Barnes, R.; Stanek, E. J. et al. (1989). How much soil do young children ingest: An epidemiologic study. Reg. Toxicol. Pharmacol. 10: 123-137.

Campbell, B. C.; Meredith, P. A.; Moore, M. R.; Watson, W. S. (1984). Kinetics of lead following intravenous administration in man. Toxicol. Ltrs. 21: 231-235.

Centers for Disease Control and Prevention (1991). Preventing lead poisoning in young children. October, 1991.

Chamberlain, A. C. (1985). Prediction of response of blood lead to airborne and dietary lead from volunteer experiments with lead isotopes. Proc. R. Soc. Lond. B. 224: 149-182.

Chamberlain, A. C.; Heard, M. J.; Little, P. et al. (1978). Investigations into lead from motor vehicles. Report of work at Environmental and Medical Sciences Division, AERE, Harwell. HL78/4122 (C.10).

Chaney, R. L.; Mielke, H. W.; Sterrett, S. B. (1988). Speciation, Mobility and Bioavailability of Soil Lead In: Environmental Geochemistry and Health Monograph Series 4. Lead in Soil: Issues and Guidelines (Eds) B. E. Davies and B. G. Wixson. Science Reviews Limited, Northwood, England. pp. 105-129.

Charney, E.; Kessler, B.; Farfel, M.; Jackson, D. (1983). Childhood lead poisoning: A controlled trial of the effect of dust-control measures on blood lead levels. NE. J. Med. 309(18): 1089-1093.

Chisholm, J. J.; Mellits, E. D.; Quaskey, S. A. (1985). The relationship between the level of lead absorption in children and the age, type, and condition of housing. Environ. Res. 38: 31-45.

Chisolm, J. J., Jr.; Harrison, H. E. (1956). Quantitative urinary coproporphyrin excretion and its relation to edathamil calcium disodium administration in children with acute lead intoxication. J. Clin Invest. 35: 1131-1138.

Clark, C. S.; Bornschein, R. L.; Ryan, J. et al. (1988). The Cincinnati Soil-Lead Abatement Demonstration Project. Presented at Lead in Soil Conference, Chapel Hill, NC, March 7-9, 1988.

Clark, C. S.; Bornschein, R. L.; Succop, P. A. et al. (1985). Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. Environ. Res. 38: 46-53.

Clarkson, T. W.; Kench, J. E. (1958). Uptake of Lead by Human Erythrocytes in vitro. Bioch 69: 432-439.

Clausing, P.; Brunekreef, B.; van Wijnen, J. H. (1987). A method for estimating soil ingestion by children. Int. Arch. Occup. Environ. Health 59: 73-82.

Cohen, J.; Marcus, A.; Elias, R. (1990). Estimating childhood multi-media lead exposure: Expanded exposure/uptake/biokinetic model. Presented at the 83rd Annual Meeting and Exhibition, Air and Water Management Assoc. Pittsburgh, PA. June 24-29, 1990. Paper 90-12.2.

Cohen, J.; Marcus, A. H.; Elias, R. W. (1991). Estimating childhood multi-media lead exposure: Expanded exposure/uptake/biokinetic model. Chemical Speciation and Bioavailability 3: 179-186.

Cohen, J.; Marcus, A.; Schwartz, J. (1988). Use of probabilistic risk assessment in development of U.S. EPA regulations on lead. Presented at Air Pollution Control Assoc meeting, New York, June 1988.

Cohen, N.; Kneip, T. J.; Rulon, V.; Goldstein, D. H. (1974). Biochemical and toxicological response of infant baboons to lead driers in paint. Environ. Health Persp. May 1974: 161-173.

Cohen, N.; Kneip, T. J.; Rulon, V.; Goldstein, D. H. (1974). Biochemical and toxicological response of infant baboons to lead driers in paint. Environ. Health Perspectives: 73: 161-173.

Cosgrove, E.; Brown, M. J.; Madigan, P. et al. (1989). Childhood lead poisoning: Case study traces source to drinking water. J. Environ. Health 52: 346-349.

Cotter-Howells, J., Thornton, I. (1991) Sources and pathways of environmental lead to children in a Derbyshire mining village. Environ. Geochem. Health 13: 127-135.

Crawford-Brown, D. J. (1983). An age-dependent model for the kinetics of uptake and removal of radionuclides from the GI tract. Health Physics 44(6): 609-622.

Davies, B. E.; Elwood, P. C.; Gallacher, J.; Ginnever, R. C. (1985). The relationships between heavy metals in garden soils and house dusts in an old lead mining area of North Wales, Great Britain. Environ. Pollution (Series B) 9: 255-266.

Davies, D. J. A.; Thornton, I.; Watt, J. M. et al. (1990). Lead intake and blood lead in two-year-old UK urban children. Sci. Total Environ. 90: 13-29.

Davis, B. K.; Jamall, I. S. (1991). Health risks in Eastern Europe associated with environmental pollution. Toxicologist 11(1): 191.

Davis, S.; Waller, P.; Buschbom, Ray; Ballou, J.; White, P. (1990). Quantitative estimates of soil ingestion in normal children between the ages of 2 and 7 years: Population-based estimates using Aluminum, Silicon, and Titanium as soil tracer elements. Arch Environ. Health 45(2) 112-122.

Dolcourt, J. L.; Finch, C.; Coleman, G. D. et al. (1981). Hazard of lead exposure in the home from recycled automobile storage batteries. Pediatrics 68(2): 225-228.

Duggan, M. J.; Inskip, M. J.; Rundle, S. A.; Moorcroft, J. S. (1985). Lead in playground dust and on the hands of schoolchildren. Sci. Total Environ. 44: 65-79.

Duggan, M. J. (1983). The uptake and excretion of lead by young children. Arch. Environ. Health 38: 246-247

Ershow, A.; and Cantor K. (1989). Total water and tap water intake in the United States: Population-based estimates of quantities and sources. Report prepared under National Cancer Institute Order #263-MD-810264. Life Sciences Research Office, Federation of American Societies for Experimental Biology. Bethesda. MD.

Farfel, M. R.; Chisolm, J. J. (1991). An evaluation of experimental practices for abatement of residential lead-based paint: Report on a pilot project. Environ. Res. 55: 199-212.

Farfel, M. R.; Chisolm, J. J. (1990). Health and environmental outcomes of traditional and modified practices for abatement of residential lead-based paint. AJPH 80(10): 1240.

Forbes, G. B.; Reina, J. C. (1972). Effect of age on gastrointestinal absorption (Fe,Sr,Pb) in the rat. J Nutr 102: 647-652.

Freeman, G. B.; Johnson, S. C.; Liao, P.; Feder, P. I.; Killinger, J. M.; Chaney, R. L.; and Bergstrom, P. D (1991). Effect of soil dose on bioavailability of lead from mining waste soil in rats. Chemical Speciation and Bioavailability 3: 121-128.

Fullmer, C. S.; Edelstein, S.; Wasserman, R. H. (1985). Lead-binding properties of intestinal calcium binding proteins. J. biol. Chem. 260: 6816-6819.

Fullmer, C. S.; Rosen, J. F. (1990). Effect of dietary calcium and lead status on intestinal calcium absorption Env. Res. 15: 91-99.

Garber, B. T.; Wei, E. (1974). Influence of dietary factors on the gastrointestinal absorption of lead. Toxicol Appl. Pharmacol. 27: 685-691.

Gardels, M. C.; Sorg, T. J. (1989). A laboratory study of the leaching of lead from water faucets. J. AWWA July 1989: 101-113.

Gartrell, M. J.; Craun, J. C.; Podrebarac, D. S.; Gunderson, E. L. (1986). Pesticides, selected elements, and other chemicals in infant and toddler total diet samples. J. Assoc. Off Anal Chem. 69(1): 123-145.

Gibaldi, M. (1982). Pharmacokinetics. Marcel Dekker, Inc. New York, New York.

Harley, N. H.; Kneip, T. H. (1985). An integrated metabolic model for lead in humans of all ages. Final report to U.S. EPA, Contract B44899, New York University Department Environmental Medicine.

Heard, M. J.; Chamberlain, A. C. (1982). Effect of minerals and food on uptake of lead from the gastrointestinal tract in humans. Hum. Toxic. 1: 411-415.

Jacobs Engineering Group Inc., Environmental Systems Division (1989). Human Health Risk Assessment Protocol for the Populated Areas of the Bunker Hill Superfund Site. Report prepared for U.S. EPA—Region X. TES IV Contract No. 68-01-7351. Moscow, Idaho.

James, H. M.; Hilburn, M. E.; Blair, J. A. (1985). Effects of meal and meal times on uptake of lead from the gastrointestinal tract in humans. Human Toxicol. 4: 401-407.

Karalekas, P. C.; Ryan, C. R.; Taylor, F. B. (1983). Control of lead, copper, and iron pipe corrosion in Boston. J. AWWA—Res. Tech., Feb 1983: 92-95.

Karam, H. S.; Beck, B. D. (1990). Current issues in determining acceptable lead concentrations in soils. Comments on Toxicology 3(6): 509-529.

Kootsey, J. M. (1989). Introduction to Computer Simulation (SCoP), National Biomedical Simulation Resource. Duke University Medical Center, Durham, NC.

Kostial, K.; Kello, D.; Jugo, S.; Rabar, I.; Maljković, T. (1978). Influence of age on metal metabolism and toxicity. Environ. Health Perspect. 25: 81-86.

Kostial, K.; Simonivic, J.; Pisonic M. (1971). Lead absorption from the intestine in newborn rats. Nature (London) 233: 564.

Lacey, R. F.; Moore, M. R.; Richards, W. N. (1985). Lead in water, infant diet and blood: The Glasgow duplicate diet study. Sci. Total Environ. 41: 235-257.

LaVelle, J. M.; Poppenga, R. H.; Thacker, B. J.; Giesy, J. P.; Weis, C. P.; Othoudt R.; Vandervoort C (1991). Bioavailability of lead in mining wastes: an oral intubation study in young swine. Chemical Speciation and Bioavailability 3: 105-111.

Laxen, D. P. H.; Raab, G. M; Fulton, M. (1987). Children's blood lead and exposure to lead in household dust and wate—a basis for an environmental standard for lead in dust. Sci. Tot. Environ. 66: 235-244.

Leggett, R. W. (1994). A model of the biokinetics of Pb in the human circulation. Submitted for publication.

Maes, E. F.; Swygert, L. A.; Paschal, D. C.; Anderson, B. S. (1991). The contribution of lead in drinking water to levels of blood lead. I. A cross-sectional study. (Submitted for publication).

Mahaffey-Six, K.; Goyer, R. A. (1972). The influence of iron deficiency on tissue content and toxicity of ingested lead in the rat. J. Lab. Clin. Med. 79: 128-136.

Mallon, R. P. (1983). A Metabolic Model of Lead Kinetics Based Upon Measured Organ Burdens During Chronic Exposure Experiments with Infant and Juvenile Baboons. Ph.D. Thesis, Department of Biology, New York University Medical Center, New York, NY.

Mallon, R. P. (1983). A metabolic model of lead kinetics based upon measured organ burdens during chronic exposure experiments with infant and juvenile baboons. Doctoral Dissertation, New York University.

Marcus, A. H. (1985a). Multicompartment kinetic model for lead: III. Lead in blood plasma and erythrocytes. Environ. Res. 36: 473-489.

Marcus, A. H. (1985b). Testing Alternative Nonlinear Kinetic Models in Compartmental Analysis. In: Mathematics and Computers in Biomedical Applications. J. Eisenfeld and C. DeLisi (Eds). Elsevier Science Publishers BV, North-Holland (IMACS).

Marcus, A. H. (1988a). Geometric standard deviation of blood lead for children in the vicinity of point sources. Report from Battelle Columbus Division, Washington Operations to U.S. EPA. Contract No. 68-02-4246.

Marcus, A. H. (1988b) Prediction of blood lead levels in East Helena children: Comparison of aggregate air lead, disaggregate, and uptake/biokinetic models. Battelle/Columbus Division, Washington Operations. Washington, D.C.

Marcus, A. H. (1989a). Uptake of lead from formula and food by infants: Reanalysis of the Ryu et al. data. Report from Battelle Columbus Division, Arlington Operations to U.S. EPA Office of Toxic Substances. Contract No. 68-02-4294.

Marcus, A. H. (1989b). Contributions to a risk assessment for lead in drinking water. Report from Battelle Arlington to U.S. EPA Office of Toxic Substances. Contract 68-D8-0115.

Marcus, A. H. (1989c). Statistical reanalyses of relationship of blood lead in Edinburgh Children to lead in dust and water. Report from Battelle Arlington Office to U.S. EPA Office of Toxic Substances. Contract No. 68-D8-0115.

Marcus, A. H. (1991a). Estimation of childhood lead burdens from newly installed faucets. Report from Battelle Columbus to U.S. EPA Office of Toxic Substances. Report No. 68-D8-0115.

Marcus, A. H. (1991b). Inter-site comparisons of environmental lead uptake. Presented at Symposium on the Bioavailability and Dietary Uptake of Lead, ECAO/USEPA. Chapel Hill, NC, September 24-27, 1990 Report from Battelle Columbus Division, Arlington Office, to U.S. EPA Office of Toxic Substances. Contract No. 68-02-4246.

Marcus, A. H. (1991c). Relationship between soil lead, dust lead, and blood lead over time: A reanalysis of the Boston lead data. Report from Battelle Columbus Division, Arlington Office, to U.S. EPA Office of Toxic Substances. Contract No. 68-02-4246.

Marcus, A. H. (1992). Use of site-specific data in models for lead risk assessment and risk management. In: *An Update of Exposure and Effects of Lead*, B. Beck (Ed), Fund. Appl. Toxicol. 18: 10-16.

Marcus, A. H.; Bernholc, A. (1990). Variability of household water lead levels in American cities. Report from Battelle Arlington Operations to U.S. EPA. Contract No. 68-D8-0115.

Marcus, A. H.; Cohen, J. (1988). Modeling the Blood Lead—Soil Lead Relationship. In: Environmental Geochemistry and Health Monograph Series 4. Lead in Soil: Issues and Guidelines. (Eds) B. E. Davies and B. G. Wixson. Science Reviews Limited, Northwood, England. pp. 161-174.

Marcus, A. H.; Elias, R. W. (1994). Estimating the contribution of lead-based paint to soil lead, dust lead, and childhood blood lead, *Lead in Paint, Soil, and Dust: Health Risks, Exposure Studies, Control Measures, Measurement Methods, and Quality Assurance, ASTM STP 1226*, Michael E. Beard and S. D. Allen Iske, Eds., American Society for Testing and Materials, Philadelphia, 1994.

Marcus, A. H.; Holtzman, A. P. (1989). Relationships between infant blood lead concentrations and lead in water or liquid diet. Report from Battelle Columbus Division to U.S. EPA Office of Drinking Water. Contract No. 68-02-4246.

Matte, T. D.; Figueroa, J. P.; Ostrowski, S.; Burr, G.; Jackson-Hunt, L.; Baker, E. L. (1991a). Lead exposure from conventional and cottage lead smelting in Jamaica. Arch. Environ. Contam. Toxicol. 21: 65-71.

Matte, T. D.; Figueroa, J. P.; Ostrowski, S.; Burr, G.; Jackson-Hunt, L.; Baker, E. L. (1991b). Relationship between soil lead levels and blood levels amorg childreb living near a lead smelter in Jamaica. Chemical Speciation and Bioavailability 3: 173-179.

Menton, R. G.; Burgoon, D. A.; Marcus, A. H. (1994). Pathways of lead contamination for the Brigham and Women's Hospital Longitudinal Lead Study, *Lead in Paint, Soil and Dust: Health Risks, Exposure Studies, Control Measures, Measurement Methods, and Quality Assurance, ASTM STP 1226*, Michael E. Beard and S.D. Allen Iske, Eds., American Society for Testing and Materials, Philadelphia, 1994.

Momcilović, B.; Kostial, K. (1974). Kinetics of lead retention and distribution in suckling rats. Arch. Environ. Health 33: 115-117.

Mordenti, J. (1986). Man versus beast: Pharmacokinetic scaling in mammals. J. Pharma. Sci. 75(11): 1028-1040.

Morton, A. P.; Partridge, S.; Blair, J. A. (1985). The intestinal uptake of lead. Chem. Brit. 21: 923-927.

Munro, I. C.; Willes, R. F.; Truelove, J. F. (1975). Absorption and tissue distribution of inorganic lead in the developing infant monkey (*Macaca irus*). Toxicol. Appl. Pharmacol. 33: 128-129.

Mushak, P. (1991). Gastro-intestinal absorption of lead in children and adults: overview of biological and biophysico-chemical aspects. Chemical Speciation and Bioavailability 3: 87-104.

Mushak, P. (1992). Defining lead as the premier environmental health issue for children in America. Environ. Res. 59: 281-309.

Mykkanen, H. M.; Wasserman, R. H. (1981). Gastrointestinal absorption of lead (^{203}Pb) in chicks: influence of lead, calcium, and age. Jutr. 111: 1757-1765.

O'Flaherty, E. J. (1986). The rate of decline of blood lead in lead industry workers during medical removal The effect of job tenure. Fund Appl. Toxicol. 6: 372-3.

O'Flaherty, E. J. (1993). Physiologically-based models for bone-seeking elements. IV. Kinetics of lead disposition in humans. Toxicol. Appld. Pharmacol. 118: 16-29.80.

Pennington, J. A. T. (1983). Revision of the total diet study food list and diets. J. Am. Dietetic Assoc. 82(2): 166-173.

Poppenga, R. H.; Thacker, B. J.; Giesy, J. P. (1990). Bioavailability of lead in mining wastes: An oral intubation study in swine. Report from Michigan State University to U.S. EPA Region VIII.

Portier, C. J.; Kaplan, N. L. (1989). Variability of safe dose estimates when using complicated models of the carcinogenic process. Fund Appl. Toxicol. 13(3): 533-544.

Proceedings of the Symposium on the Bioavailability and Dietary Exposure of Lead, (1991) September 1990, Chapel Hill, North Carolina. Chemical Speciation and Bioavailability, Volume 3, No 3-4. ISBN 0-946682-10-0.

Raab, G. M.; Laxen, D. P. H.; Fulton, M. (1987). Lead from dust and water as exposure sources for children. Environ. Geochem. Health 9(3-4): 80-85.

Rabinowitz, M. B. (1987). Stable isotope mass spectrometry in childhood lead poisoning. Biological Trace Element Research. 12: 223-229.

Rabinowitz, M. B. (1987). Stable isotope mass spectrometry in childhood lead poisoning. Biological Trace Element Research. 12: 223-229.

Rabinowitz, M. B.; Kopple, J. D.; Wetherill, G. W. (1976). Kinetic analysis of lead metabolism in healthy humans. J. Clin. Invest. 58: 260-270.

Rabinowitz, M. B.; Kopple, J. D.; Wetherill, G. W. (1980). Effect of food intake and fasting on gastrointestinal lead absorption in humans. Am. J. Clin. Nutr. 33: 1784-1788.

Rabinowitz, M.; Leviton, A.; Bellinger, D. (1984). Home refinishing, lead paint, and infant blood lead levels. Amer. J. Public Health. 75: 403-404.

Rabinowitz, M.; Leviton, A.; Needleman, H.; Bellinger, D.; Waternaux, C. (1985). Environmental correlates of infant blood lead levels in Boston. Environ. Res. 38: 96-107.

Revicki, D. A.; Blixhamser, A.; Hersey, J.; Marcus, A. (1991). The cost-effectiveness of alternative lead abatement strategies. Report from Battelle to CDC/CEHIC.

Ryu, J. E.; Ziegler, E. E.; Nelson, S. E.; Fomon, S. J. (1983). Dietary intake of lead and blood lead concentration in early infancy. Am. J. Dis. Child. 137: 886-891.

Sawyer, M.; Kearney, T.; Spector, S. et al. (1985). Lead intoxication in children—Interdepartmental Conference University of California, San Diego (Specialty Conference). West J. Med. 143: 357-364.

Schilling, R. J.; Bain, R. P. (1988). Prediction of children's blood lead levels on the basis of household-specific soil lead levels. Am. J. Epidemiology 128(1): 197-205.

Schock, M. R.; Neff, C. H. (1988). Trace metal contamination from brass fittings. J. AWWA 80(11): 47-56.

Sherlock, J. C.; Quinn, M. J. (1986). Relationship between blood lead concentrations and dietary lead intake in infants: The Glascow Duplicate Diet Study 1979-1980. Food Additives and Contaminants 3: 167-175.

Simmons, C. M. (1989). City of Raleigh's experience in the pilot public education program for lead in drinking water. Raleigh, North Carolina.

Stark, A. D.; Quah, R. F.; Meigs, J. W.; DeLouise, E. R. (1982). The relationship of environmental lead to blood-lead levels in children. Environ. Res. 27: 372-383.

Steele, M. J.; Beck, B. D.; Murphy, B. L. (1990). Assessing the contribution from lead in mining wastes to blood lead. Reg. Toxicol. Pharm., 11: 158-170.

Succop, P. A.; O'Flaherty; Bornschein, R. L. et al. (1987). A kinetic model for estimating changes in the concentration of lead in the blood of young children. In: International Conference: Heavy Metals in the Environment, (Eds) Lindberg, S. E.; Hutchinson, T. C.; New Orleans, September 1987. (EP Consultants Ltd., Edinburgh, pp. 289-291).

Thornton, E.; Daview, D. J. A.; Watt, J. M.; Quinn, M. J. Lead exposure in young children from dust and soil in the United Kingdom. Environ. Health Persp. 89: 55-60.

Trotter, R. T. (1990). The cultural parameters of lead poisoning: A medical anthropologist's view of intervention in environmental lead exposure. Environ. Health Persp. 89: 79-84.

Tukey, J. W. (1977) *Exploratory Data Analysis*. Addison-Wesley Publishing Co., Reading MA.

U.S. Department of Housing and Urban Development (1990). Comprehensive and workable plan for the abatement of lead-based paint in privately owned housing. Office of Policy Development and Urban Research. PB91-193953.

U.S. Environmental Protection Agency (1986). Air Quality Criteria for Lead Volume I-IV. Environmental Criteria and Assessment Office, Office of Research and Development, RTP, NC. EPA 600/8-83-028 a-d

U.S. Environmental Protection Agency (1989a). Review of the National Ambient Air Quality Standards for Lead Exposure Analysis Methodology and Validation. U.S. EPA Office of Air Quality Planning and Standards. RTP, NC. EPA-450/2-89/011.

U.S. Environmental Protection Agency (1989b). Guidance Concerning Soil Lead Cleanup Levels at Superfund Sites. Directive #9355.4-02. Office of Solid Waste and Emergency Response. September 1989.

U.S. Environmental Protection Agency (1989c). Exposure Factors Handbook. U.S. EPA Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-89/043.

U.S. Environmental Protection Agency (1990a). Technical Support Document on Lead. Document prepared for Office of Solid Waste and Emergency Response by U.S. EPA/ECAO Cincinnati, OH. ECAO-CIN-757.

U.S. Environmental Protection Agency (1990b). Report of the Clean Air Scientific Advisory Committee on its Review of the OAQPS Lead Staff Paper and the ECAO Air Quality Criteria Document Supplement. EPA-SAB-CASAC-90-002. January 1990.

U.S. Environmental Protection Agency (1990c). Risk Assessment Data Evaluation Report (RADER), by TerraGraphics Environmental Engineering under Contract #68-W9-0008, WA #C10012, October 1990.

U.S. Environmental Protection Agency (1991a). Three City Urban Soil-Lead Demonstration Project—Midterm Project Update. U.S. EPA ECAO/HEARD. 21S2001.

U.S. Environmental Protection Agency (1991b). National Air Quality and Emissions Trends Report 1989 Report prepared by U.S. EPA OAQPS, RTP, NC. EPA-450/4-91-003.

U.S. Environmental Protection Agency (1991c). National Primary Drinking Water Regulation for Lead (NPDWR). Federal Register 56-26460. June 7, 1991.

U.S. Environmental Protection Agency (1991d). Sampling Manual for Site-Specific Data Collection for the Lead Integrated Biokinetic Uptake Model. Report to U.S. EPA from Cadmus Assoc. (in preparation).

U.S. Environmental Protection Agency (1992a). An SAB Report: Review of the Uptake Biokinetic Model for Lead. EPA-SAB-IAQC-92016. March 1992.

U.S. Environmental Protection Agency (1992b). Guidance for Data Useability in Risk Assessment (Part A). Directive #9285.7-09A. Office of Emergency and Remedial Response. April 1992.

U.S. Environmental Protection Agency (1993). Urban Soil Lead Abatement Demonstration Project, Volume I: Integrated Report, Office of Research and Development. EPA/600/AP-93/001a, July 1993.

van Wijnen, J. H.; Clausing, P.; Brunekreef, B. (1990). Estimated soil ingestion by children. Environ. Res. 51: 147-162.

von Lindern, I. (1991). Projected Blood Lead Distribution for Proposed Soil Cleanup Guidelines—Bunker Hill Site Residential Soils Feasibility Study (Technical Memorandum from TerraGraphics Environmental Engineering to Sally Martyn, U.S. EPA Region X).

Walter, S. D.; Yankel, A. J.; von Lindern, I. H. (1980). Age-specific risk factors for lead absorption in children. Arch. Environ. Health 35:53-58.

Weis, C. P.; Poppenga, R. H.; Thacker, P. J. (1994). Design of pharmacokinetic and bioavailability studies of lead in an immature swine model. In: Lead in paint, soil, and dust: Health risks, exposure studies, control measures, measurement methods, and quality assurance. Beard, M.E.; Iske, S.A., Eds. ASTM 1226. Philadelphia: American Society for Testing and Materials.

Weis, C. P.; LaVelle, J. M. (1991). Characteristics to consider when choosing an an animal model for the study of lead bioavailability. In: Proceedings of the Symposium on the Bioavailability and Dietary Exposure of Lead, Chemical Speciation and Bioavailability 3: 113-119.

Weitzman, M.; Aschengrau, A.; Bellinger, D.; Jones, R.; Hamlin, J. S.; Beiser, A. (1993). Lead-contaminated soil abatement and urban children's blood lead levels. J. Amer. Med. Assoc. 269: 1647-1654.

West, J. R.; Smith, H. W.; Chasis, H. (1948). Glomerular filtration rate, effective renal blood flow, and maximal tubular excretory capacity in infancy. J. Pediatr. 32: 10-18.

Whitfield, R. G.; Cohen, J.; Marcus, A. H.; Zaragoza, L. J. (1989). Using data and judgment to estimate blood-lead distributions among children from soil-lead levels and other environmental factors. Unpublished report for Argonne National Laboratory, Energy and Environmental Systems Division.

Woodward-Clyde (1991). Demographics workplan California Gulch Study Area Leadville, Colorado. Prepared for Asarco by WoodwardClyde. Project No. 22443E-33441. July 1991.

Yaffe, Y. et al. (1983). Identification of lead sources in California children using the stable isotope ratio technique. Arch. Environ. Health 38(4): 237-245.

Yankel, A. J.; von Lindern, I. H.; Walter, S. D. (1977). The Silver Valley lead study: the relationship of childhood lead poisoning and environmental exposure. J. Air Pollut. Control Assoc. 27: 763-767.

Ziegler, E. E.; Edwards, B. B.; Jensen, R. L.; Mahaffey, K. R.; Fomon, S. J. (1978). Absorption and retention of lead by infants. Pediatr. Res. 12: 29-34.

APPENDIX A: HOW TO CALCULATE THE GEOMETRIC STANDARD DEVIATION FROM BLOOD LEAD DATA, IF YOU MUST

A.1 A DIRECT METHOD FOR CALCULATING THE GEOMETRIC STANDARD DEVIATION

One of the simplest approaches to calculating a GSD from a sample of blood lead and environmental lead data is based on the idea that children with similar environmental lead exposures will have similar geometric mean blood lead levels. For children of a given age with similar soil lead (denoted PbS), dust lead (denoted PbD), and other lead exposures, we can reasonably characterize the variability in blood lead level (denoted GSD) calculated with respect to the actual geometric mean blood lead level of this group of children (denoted GMB) without modelling blood lead levels. The procedure shown here is the simplest procedure we have found, but even with this procedure, the user must be prepared to do a great deal of statistical calculation. We will illustrate how an empirical GSD may be calculated from data after we describe the procedure:

STEP 1: Divide the data set into subgroups, where each group has children of a given age, with soil lead levels in a given interval, dust lead levels in a given interval, and with distinct levels of other important variables. Each such group corresponds to a "box" or cell of soil and dust lead levels, and levels of other variables if used.

STEP 2: From each individual blood lead (denoted PbB) in each cell, calculate ln(PbB), where ln denotes the natural logarithm.

STEP 3: Within each cell, calculate the mean and the standard deviation of the ln(PbB) values. Then, for that cell,

GMB = exp(mean of ln(PbB) values within the cell)
GSD = exp(standard deviation of ln(PbB) values within the cell)

where exp denotes the process of calculating the exponential function of the indicated quantity. Exponential and natural logarithm functions are available in most statistical packages for microcomputers and on most scientific calculators.

STEP 4: Calculate the inter-individual GSD for this neighborhood by finding the median or middle value of the GSD values in the sample. The median is found by ordering the GSD values from all cells from smallest to

largest. If the number of GSD values is odd, the median is the middle value; if sample size is an even number, find the average of the two middle values. Since the number of observations in each box or cell is different, each GSD should be counted a number of times according to the number of degrees of freedom (cell count minus one) for that GSD.

STEP 5: Users with some statistical background may wish to examine the within-cell GSD's for patterns based on the data, such as by plotting GSD against GMB or against the within-cell value of age, PbS, PbD, or other stratifying variables.

STEP 6: Users with more statistical background may wish to use other approaches to calculating a "typical" GSD, such as by calculating a mean or pooled variance of the within-cell variances of ln(PbB). We would caution such users that the data should be carefully evaluated for outliers, either in raw PbB values or in the calculated GSD. One convenient approach for visual detection of outliers is a normal probability of within-cell variances after a variance-stabilizing transformation such as the cube root of the within-cell variance of ln(PbB).

EXAMPLE: In a sample of 166 children from the Midvale, Utah study of 1989 (Bornschein et al., 1990), we found that the estimation of blood lead levels could be considerably improved by determining whether or not the children lived in houses in which paint had recently been removed. There is substantial evidence that inadequately controlled lead paint abatement may increase blood levels in resident young children by 2 to 4 ug/dL on average in the first 6 to 12 months after paint removal (Rabinowitz et al., 1984; Marcus et al., 1991; Menton et al., 1993). Interviews with the family provided such information for 162 of the 166 children, which was used as an additional stratifying variable.

The worksheet for determining subgroups are shown in Table A-1. Each table gives the blood leads of children of a given age, divided by whether or not there was recent paint removal in the residence. Within each table, the children are divided according as the PbS and PbD values at their residence. Each cell in the table corresponds to intervals of 250 ppm of PbS and 250 ppm of PbD. The soil lead levels were averages of non-missing values of perimeter, bare area, play area, and garden soils. It should be noted that data for most of the cells are not available with such detailed sub-division of the data set, and that at higher soil and dust lead concentrations, there is usually only one observation per cell. There was only one case in which two children from the same family had the same age, in years, and analyses without this duplication would produce very similar results. Otherwise, all blood lead levels (denoted PbB) within each cell come from different families. This is believed to

TABLE A-1. CELLS OF BLOOD LEAD LEVELS IN 165 MIDVALE CHILDREN, BY PAINT REMOVAL STATUS, AGE, AND INTERVALS OF 250 μg/g IN SOIL AND DUST LEAD[1]

Paint Removal	Age	Soil Pb	Dust Pb	Blood Lead (μg/dL) Smallest →→Largest					
.	0	375	375	5.5	.	.	.	.	.
.	2	.	625	6.	.	.	.	.	.
.	2	375	125	4.	.	.	.	.	.
.	3	625	625	3.	.	.	.	.	.
.	4	125	125	6.5	.	.	.	.	.
0	0	125	.	3.	6.	.	.	.	.
0	0	125	125	1.	.	.	.	.	.
0	0	125	375	0.5	.	.	.	.	.
0	0	375	125	3.	4.5	.	.	.	.
0	0	375	375	5.5	.	.	.	.	.
0	0	375	1125	3.	7.	.	.	.	.
0	0	375	1375	3.5	.	.	.	.	.
0	0	1125	875	13.5	.	.	.	.	.
0	1	.	625	5.5	.	.	.	.	.
0	1	125	375	2.5	3.	.	.	.	.
0	1	375	375	4.	5.5	7.	.	.	.
0	1	375	625	4.5	.	.	.	.	.
0	1	375	875	3.5	.	.	.	.	.
0	1	625	375	3.	7.	8.	10.	.	.
0	1	625	625	3.5	6.	6.	.	.	.
0	1	875	625	3.	.	.	.	.	.
0	1	875	1125	6.	.	.	.	.	.
0	1	1125	875	1.	10.5	.	.	.	.
0	1	1375	1125	6.	.	.	.	.	.
0	1	1625	625	3.	.	.	.	.	.
0	2	.	375	4.	6.	.	.	.	.

TABLE A-1 (cont'd). CELLS OF BLOOD LEAD LEVELS IN 165 MIDVALE CHILDREN, BY PAINT REMOVAL STATUS, AGE, AND INTERVALS OF 250 μg/g IN SOIL AND DUST LEAD[1]

Paint Removal	Age	Soil Pb	Dust Pb	Blood Lead (μg/dL) Smallest →→Largest					
0	2	.	625	7.	.	.	.	.	.
0	2	125	.	5.	.	.	.	.	.
0	2	125	125	2.5	5.5	5.5	8.	12.	.
0	2	125	625	6.	.	.	.	.	.
0	2	375	375	1.5	.	.	.	.	.
0	2	375	1125	4.5	.	.	.	.	.
0	2	625	375	14.5	.	.	.	.	.
0	2	625	625	4.	7.	11.5	.	.	.
0	2	875	1125	5.5	.	.	.	.	.
0	2	1125	.	13.	.	.	.	.	.
0	2	1125	625	9.5	.	.	.	.	.
0	2	1125	875	19.	.	.	.	.	.
0	3	.	625	5.	.	.	.	.	.
0	3	125	125	2.5	.	.	.	.	.
0	3	125	375	2.	7.5	.	.	.	.
0	3	375	125	6.5	.	.	.	.	.
0	3	375	375	3.	4.	.	.	.	.
0	3	375	1375	13.	.	.	.	.	.
0	3	1125	625	16.5	.	.	.	.	.
0	3	1375	1125	5.	.	.	.	.	.
0	4	.	375	2.	.	.	.	.	.
0	4	125	125	4.	7.5	.	.	.	.
0	4	125	375	5.5	6.	.	.	.	.
0	4	375	.	2.	.	.	.	.	.
0	4	375	625	1.5	7.	.	.	.	.

TABLE A-1 (cont'd). CELLS OF BLOOD LEAD LEVELS IN 165 MIDVALE CHILDREN, BY PAINT REMOVAL STATUS, AGE, AND INTERVALS OF 250 μg/g IN SOIL AND DUST LEAD[1]

Paint Removal	Age	Soil Pb	Dust Pb	Blood Lead (μg/dL) Smallest → Largest					
0	4	625	375	5.	.	.	.	.	.
0	4	875	625	7.5	.	.	.	.	.
0	4	1125	2375	5.	.	.	.	.	.
0	4	2125	875	8.	.	.	.	.	.
0	5	125	125	2.	4.5	8.5	.	.	.
0	5	125	375	2.5	3.5	10.	.	.	.
0	5	375	375	4.	6.	.	.	.	.
0	5	625	375	5.	.	.	.	.	.
0	5	625	625	4.	.	.	.	.	.
0	5	625	1375	4.5	.	.	.	.	.
0	5	1125	875	13.5	.	.	.	.	.
1	0	125	125	3.	16.5	.	.	.	.
1	0	125	375	0.5	1.5	3.5	5..	6.	.
1	0	375	375	8.5	.	.	.	.	.
1	0	375	875	5.	.	.	.	.	.
1	1	.	375	8.	22.5	.	.	.	.
1	1	125	125	5.5	.	.	.	.	.
1	1	125	375	5.5	.	.	.	.	.
1	1	375	375	3.5	.	.	.	.	.
1	1	375	625	5.5	.	.	.	.	.
1	1	375	875	16.5	.	.	.	.	.
1	1	625	125	2.5	.	.	.	.	.
1	1	625	375	9.	9.	.	.	.	.
1	1	875	625	6.	.	.	.	.	.
1	1	1125	1375	5.5	.	.	.	.	.

TABLE A-1 (cont'd). CELLS OF BLOOD LEAD LEVELS IN 165 MIDVALE CHILDREN, BY PAINT REMOVAL STATUS, AGE, AND INTERVALS OF 250 μg/g IN SOIL AND DUST LEAD[1]

Paint Removal	Age	Soil Pb	Dust Pb	Blood Lead (μg/dL) Smallest →→Largest					
1	2	.	.	8.5	.	.	.	.	
1	2	125	125	3.	4.	4.5	5.5	.	.
1	2	125	375	2.5	3.5	5.	5.	5.5	19.5
1	2	375	.	6.	.	.	.	.	.
1	2	375	375	3.	10.	.	.	.	.
1	2	375	625	8.5	.	.	.	.	.
1	2	625	625	6.5	.	.	.	.	.
1	2	1875	625	10.5	.	.	.	.	.
1	3	.	.	8.5	.	.	.	.	.
1	3	.	625	4.	.	.	.	.	.
1	3	125	375	4.5	.	.	.	.	.
1	3	375	625	5.5	5.5	8.	.	.	.
1	3	375	875	4.	.	.	.	.	.
1	3	625	875	2.	.	.	.	.	.
1	3	875	3625	2.	.	.	.	.	.
1	3	1625	1375	15.5	.	.	.	.	.
1	3	1875	625	7.5	.	.	.	.	.
1	4	.	375	3.5	18.	.	.	.	.
1	4	.	625	3.5	.	.	.	.	.
1	4	125	125	4.5	5.	5.	5.	5.	.
1	4	125	375	2.	3.5	4.	8.	.	.
1	4	375	.	7.	.	.	.	.	.
1	4	875	625	9.	.	.	.	.	.
1	4	875	1125	7.5	.	.	.	.	.
1	4	1625	1375	9.5	.	.	.	.	.

TABLE A-1 (cont'd). CELLS OF BLOOD LEAD LEVELS IN 165 MIDVALE CHILDREN, BY PAINT REMOVAL STATUS, AGE, AND INTERVALS OF 250 μg/g IN SOIL AND DUST LEAD[1]

Paint Removal	Age	Soil Pb	Dust Pb	Blood Lead (μg/dL) Smallest →→Largest					
1	4	3125	1625	13.	.	.	.	.	.
1	5	.	.	4.5	.	.	.	.	.
1	5	125	125	4.	5.	.	.	.	.
1	5	125	375	4.	7.5	.	.	.	.
1	5	375	375	1.5	.	.	.	.	.
1	5	625	375	6.5	.	.	.	.	.
1	5	1875	625	5.5	.	.	.	.	.

[1] An isolated decimal point denotes a missing value.

give a much more valid estimate of variability than within-family GSD's for children of different ages, but similar genetic and non-lead environmental factors and similar family behavior patterns.

The statistics for GMB and GSD were calculated as described in Step 3, for each cell where enough data were available (at least 2 PbB values in order to calculate GSD). The results are shown in Table A-2. The PbS and PbD values are the cell midpoints, and provide convenient plot points. Some of the GSD values are very high, as for the cell whose two values are PbB = 1.5 and 10 μg/dL.

The distribution of GSD values for all cells is shown in Table A-3, in the form of a "stem-and-leaf" plot (Tukey, 1977). No weighting scheme has been applied. Many users would prefer a weighted GSD where the number of observations in each cell is taken into account. This can be done by counting each GSD estimate as representing the number of degrees of freedom (denoted DF) in the GSD estimate. In this application, DF = N − 1, where N is the number of PbB values in the cell. A DF-weighted stem-and-leaf plot is shown in Table A-4. In the unweighted case, the median GSD = 1.694 may be taken as a representative value for this community. In the weighted DF case, a somewhat larger median GSD = 1.768 may be used.

TABLE A-2. GEOMETRIC MEAN AND GEOMETRIC STANDARD DEVIATION OF BLOOD LEADS IN CELLS OR GROUPS, BY PAINT REMOVAL STATUS, AGE, AND INTERVALS OF 250 μg/g IN SOIL AND DUST LEAD[1]

Paint Removal	Age (Years)	Soil Lead (μg/g)	Dust Lead (μg/g)	N	Geometric Mean Blood Lead (μg/dL)	GSD
.	0	375	375	1	5.5	.
.	2	.	625	1	6.	.
.	2	375	125	1	4.	.
.	3	625	625	1	3.	.
.	4	125	125	1	6.5	.
0	0	125	.	2	4.243	1.633
0	0	125	125	1	1.	.
0	0	125	375	1	0.5	.
0	0	375	125	2	3.674	1.332
0	0	375	375	1	5.5	.
0	0	375	1125	2	4.583	1.821
0	0	375	1375	1	3.5	.
0	0	1125	875	1	13.5	.
0	1	.	625	1	5.5	.
0	1	125	375	2	2.739	1.138
0	1	375	375	3	5.360	1.324
0	1	375	625	1	4.5	.
0	1	375	875	1	3.5	.
0	1	625	375	4	6.402	1.693
0	1	625	625	3	5.013	1.365
0	1	875	625	1	3.	.
0	1	875	1125	1	6.	.
0	1	1125	875	2	3.240	5.273
0	1	1375	1125	1	6.	.

TABLE A-2 (cont'd). GEOMETRIC MEAN AND GEOMETRIC STANDARD DEVIATION OF BLOOD LEADS IN CELLS OR GROUPS, BY PAINT REMOVAL STATUS, AGE, AND INTERVALS OF 250 μg/g IN SOIL AND DUST LEAD[1]

Paint Removal	Age (Years)	Soil Lead (μg/g)	Dust Lead (μg/g)	N	Geometric Mean Blood Lead (μg/dL)	GSD
0	1	1625	625	1	3.	.
0	2	.	375	2	4.899	1.332
0	2	.	625	1	7.	.
0	2	125	.	1	5.	.
0	2	125	125	5	5.055	2.013
0	2	125	625	1	6.	.
0	2	375	375	1	1.5	.
0	2	375	1125	1	4.5	.
0	2	625	375	1	14.5	.
0	2	625	625	3	6.854	1.696
0	2	875	1125	1	5.5	.
0	2	1125	.	1	13.	.
0	2	1125	625	1	9.5	.
0	2	1125	875	1	19.	.
0	3	.	625	1	5.	.
0	3	125	125	1	2.5	.
0	3	125	375	2	3.873	2.546
0	3	375	125	1	6.5	.
0	3	375	375	3	4.762	1.768
0	3	375	1375	1	13.	.
0	3	1125	625	1	16.5	.
0	3	1375	1125	1	5.	.
0	4	.	375	1	2.	.

TABLE A-2 (cont'd). GEOMETRIC MEAN AND GEOMETRIC STANDARD DEVIATION OF BLOOD LEADS IN CELLS OR GROUPS, BY PAINT REMOVAL STATUS, AGE, AND INTERVALS OF 250 μg/g IN SOIL AND DUST LEAD[1]

Paint Removal	Age (Years)	Soil Lead (μg/g)	Dust Lead (μg/g)	N	Geometric Mean Blood Lead (μg/dL)	GSD
0	4	125	125	2	5.477	1.560
0	4	125	375	2	5.745	1.063
0	4	375	.	1	2.	.
0	4	375	625	2	3.240	2.972
0	4	625	375	1	5.	.
0	4	875	625	1	7.5	.
0	4	1125	2375	1	5.	.
0	4	2125	875	1	8.	.
0	5	125	125	3	4.245	2.065
0	5	125	375	3	4.440	2.061
0	5	375	375	2	4.899	1.332
0	5	625	375	1	5.	.
0	5	625	625	1	4.	.
0	5	625	1375	1	4.5	.
0	5	1125	875	1	13.5	.
1	0	125	125	2	3.	16.5
1	0	125	375	5	0.5	1.5
1	0	375	375	1	8.5	.
1	0	375	875	1	5.	.
1	1	.	375	2	8.	22.5
1	1	125	125	1	5.5	.
1	1	125	375	1	5.5	.
1	1	375	375	1	3.5	.

TABLE A-2 (cont'd). GEOMETRIC MEAN AND GEOMETRIC STANDARD DEVIATION OF BLOOD LEADS IN CELLS OR GROUPS, BY PAINT REMOVAL STATUS, AGE, AND INTERVALS OF 250 μg/g IN SOIL AND DUST LEAD[1]

Paint Removal	Age (Years)	Soil Lead (μg/g)	Dust Lead (μg/g)	N	Geometric Mean Blood Lead (μg/dL)	GSD
1	1	375	625	1	5.5	.
1	1	375	875	1	16.5	.
1	1	625	125	1	2.5	.
1	1	625	375	1	9.	9.
1	1	875	625	2	6.	.
1	1	1125	1375	1	5.5	.
1	2	.	.	1	8.5	.
1	2	125	125	4	3.	4.
1	2	125	375	6	2.5	3.5
1	2	375	.	1	6.	.
1	2	375	375	2	3.	10.
1	2	375	625	1	8.5	.
1	2	625	625	1	6.5	.
1	2	1875	625	1	10.5	.
1	3	.	.	1	8.5	.
1	3	.	625	1	4.	.
1	3	125	375	1	4.5	.
1	3	375	625	3	5.5	5.5
1	3	375	875	1	4.	.
1	3	625	875	1	2.	.
1	3	875	3625	1	2.	.
1	3	1625	1375	1	15.5	.
1	3	1875	625	1	7.5	.

TABLE A-2 (cont'd). GEOMETRIC MEAN AND GEOMETRIC STANDARD DEVIATION OF BLOOD LEADS IN CELLS OR GROUPS, BY PAINT REMOVAL STATUS, AGE, AND INTERVALS OF 250 μg/g IN SOIL AND DUST LEAD[1]

Paint Removal	Age (Years)	Soil Lead (μg/g)	Dust Lead (μg/g)	N	Geometric Mean Blood Lead (μg/dL)	GSD
1	4	.	375	2	3.5	18.
1	4	.	625	1	3.5	.
1	4	125	125	5	4.5	5.
1	4	125	375	4	2.	3.5
1	4	375	.	1	7.	.
1	4	875	625	1	9.	.
1	4	875	1125	1	7.5	.
1	4	1625	1375	1	9.5	.
1	4	3125	1625	1	13.	.
1	5	.	.	1	4.5	.
1	5	125	125	2	4.	5.
1	5	125	375	2	4.	7.5
1	5	375	375	1	1.5	.
1	5	625	375	1	6.5	.
1	5	1875	625	1	5.5	.

A.2 A MORE SOPHISTICATED STATISTICAL METHOD FOR ESTIMATING THE GEOMETRIC STANDARD DEVIATION

The GSD actually represents the residual variability in the logarithm of the predicted blood lead level. A direct regression method that is an overly simplified approximation to the IEUBK model at steady state exposure may be useful in deriving a residual GSD from a blood lead and environmental lead study. The method is based on the concepts that: (1) the IEUBK model at low to moderate steady-state exposure yields predicted blood leads that are

TABLE A-3. STEM AND LEAF PLOT OF GEOMETRIC STANDARD DEVIATION FOR MIDVALE CHILDREN[1,2]

MINIMUM:	1.048	
LOWER QUARTILE:	1.332	
MEDIAN:	1.694	
UPPER QUARTILE:	2.071	
MAXIMUM:	5.273	
1		0011
1	H	22333333
1		55
1	M	6667
1		88
2	H	00000
2		3
2		5
2		7
2		9
OUTSIDE VALUES		
3		13
5		2

[1]N = 32 groups, unweighted.
[2]76 groups with missing values excluded from plot.

approximately linear functions of PbS and PbD, with age-dependent regression coefficients; (2) the linear model should be fitted in a logarithmic form so as to estimate relative variability. In order to use the model, it is necessary to create indicator variables for the age of the child in the study. These are:

TABLE A-4. STEM AND LEAF PLOT OF
GEOMETRIC STANDARD DEVIATION FOR MIDVALE CHILDREN[1]
(Weighted by Degrees of Freedom)

MINIMUM:	1.048	
LOWER QUARTILE:	1.332	
MEDIAN:	1.768	
UPPER QUARTILE:	2.061	
MAXIMUM:	5.273	
1		0000011
1	H	2222233333333
1		55
1	M	66666677
1		8888
2	H	00000000000000
2		3
2		5
2		7777
2		9
OUTSIDE VALUES		
3		13
5		2

[1]N = 58 groups, weighted by degrees of freedom.

AGE0 = 1 if the child is age 0 to 11 months; AGE0 = 0 if not;
AGE1 = 1 if the child is age 12 to 23 months; AGE1 = 0 if not;
AGE2 = 1 if the child is age 24 to 35 months; AGE2 = 0 if not;
AGE3 = 1 if the child is age 36 to 47 months; AGE3 = 0 if not;
AGE4 = 1 if the child is age 48 to 59 months; AGE4 = 0 if not;
AGE5 = 1 if the child is age 60 to 71 months; AGE5 = 0 if not;

AGE6 = 1 if the child is age 72 to 83 months; AGE6 = 0 if not;

and so on. Then the model that may be fitted, using all of the children in the data set for which observed or imputed PbS and PbD values are available, using a nonlinear regression program for parameter estimation, is given by

$$\ln(PbB) = \ln(A0*AGE0 + A1*AGE1 + A2*AGE2 + A3*AGE3 + \ldots + PbS * (B0*AGE0 + B1*AGE1 + B2*AGE2 + B3*AGE3 + \ldots) + + PbD * (C0*AGE0 + C1*AGE1 + C2*AGE2 + C3*AGE3 + \ldots) + + X * (D0*AGE0 + D1*AGE1 + D2*AGE2 + D3*AGE3 + \ldots))$$

Here, X represents other predictive covariates for blood lead. In the Midvale example, X = RMVPAINT = 1 if paint has recently been removed from the premises, and X = 0 if not. In other applications, it may be useful to use water lead or air lead levels as an additional predictor. X may be omitted if necessary. In many applications, the regression parameters may be set equal for some ages. For example, if blood leads stabilize for ages 3 to 5 years, we may set A3 = A4 = A5, B3 = B4 = B5, C3 = C4 = C5, etc. Since the age-dependence of soil and dust lead exposure may differ somewhat from one site to another, depending on climate or other factors, no general prescription for how to carry out such analyses may be given.

When a non-linear regression model is fitted to the data by use of a program that estimates non-linear parameters, it is then possible to calculate the residual standard deviation S for the model, so that

S = standard deviation of ln(observed PbB / predicted PbB)

GSD = exp(S).

For the Midvale example described in this Appendix, we find that for N = 143 children with no missing data for PbD, PbS, or RMVPAINT, S = 0.5701 on the natural log scale, thus GSD = 1.768. The approximate relative standard error of S^2 is $(2 / (N - p))^{0.5}$, where p is the number of nonlinear parameters estimated from the data. With p = 12 parameters (ages 2 to 5 years were grouped), we have $(2 / (143 - 12))^{0.5} = 0.1236$ relative standard deviation for the variance. An approximate 95% confidence interval for the true value of S^2 has a lower bound $(1 - 2 * (2 / (N - p))^{0.5})$, and an upper bound $(1 + 2 * (2 / (N - p))^{0.5})$, times S^2. For Midvale, the limits are

$$(1 - 2 * 0.1236) * (0.5701)^2 = 0.2447$$

$$(1 + 2 * 0.1236) * (0.5701)^2 = 0.4053$$

Thus the lower and upper bounds for S are 0.4947 to 0.6366, and for GSD = exp(S) the confidence limits are 1.640 to 1.890.

APPENDIX B: SUMMARY OF REVISIONS TO LEAD UPTAKE BIOKINETIC MODEL SOFTWARE VERSIONS

TABLE B-1. SUMMARY OF REVISIONS TO LEAD UPTAKE/BIOKINETIC MODEL SOFTWARE FROM LEAD 0.2 TO LEAD 0.4

Item	Lead 0.2 (September, 1989)	Lead 0.4 (September, 1990)
GI tract absorption model	Default is linear "passive" model.	Default is non-linear "active passive" model
Drinking water defaults	Composite = 9 μg/L First flush = 0 μg/L Flushed = 9 μg/L Fountain = 0 μg/L % First flush = 0 % Flushed = 0 % Fountain = 0	Composite = 4 μg/L First flush = 4 μg/L Flushed = 1 μg/L Fountain = 10 μg/L % First flush = 50 % Flushed = 35% % Fountain = 15% New defaults were provided EPA/ODW)
Diet defaults	Dietary lead intakes are based on FDA surveys completed in 1986. Default values range from 22-34 μg/day for ages 0-7.	Dietary lead intakes are based on FDA surveys completed in 1988. Default values range from 3.8-7.5 μg/day for ages 0-7.
Probability density function	P(x) = (1/(2.51)(GSD)); exp((-1/2)((lnx-lnGM)/ lnGS D)2)	P(x) = (1/(2.51)(x)(lnGSD)); exp((-1/2)((lnx lnGM)/ lnGSD)2)
Plot of probability density function	Ordinate of "Bell-shaped plot" labelled "probability".	Ordinate of "bell-shaped plot" labelled "probability density function f(blood lead)" numbers removed from ordinate. Probability is computed as the integral of the function over a specified ranged. This is graphically illustrated in the "S shaped" probability percent plot.
Calculation of blood lead in newborn (BPbn)	Fixed at 6.4 μg/dL. BPbn = BPbm ; k, where BPbm is the maternal blood lead (7.5 μg/dL) and k is a constant (0.85).	User option: 1) As in Lead 0.2 and option to select maternal blood lead. 2) "Fetal" model, in which maternal blood lead is calculated based on user-defined exposure data and a maternal biokinetic model, and newborn blood lead is calculated from biokinetic model
Soil/Dust primary data entry screen	Menu includes "Change GI Absorption Method"	Menu revised to "Change GI Method/Bioavailability". If "yes" is selected, a window containing the following message appears over the secondary data entry screen: "Bioavailability of soil lead may vary depending on the lead source. For example, lead from mine wastes may have a lower bioavailability than lead from smelters. Differences in bioavailability are thought to reflect differences in gastrointestinal absorption of specific lead species and particle sizes, which vary depending on the source. The following data entry screen allows the user to make adjustments in the gastrointestinal absorption coefficients to account for site specific information on bioavailability

TABLE B-2. SUMMARY OF REVISIONS TO LEAD UPTAKE BIOKINETIC MODEL SOFTWARE FROM LEAD 0.4 TO LEAD 0.5

Parameter or Feature	Lead 0.4 (September, 1990)	Lead 0.5 (December, 1990)
(Non-linear GI Absorption Model	Volume of GI-tract calculated, VOI = (VOI)/(OF); where VOI is the adult GI-tract volume (liters) and OF is the age-dependent allometric scaling factor (OF < 1 for children). Default value for Km, 100 µg/L.	Volume of GI tract calculated, VOI = (VOI)(OF). Default value for Km, 100 mg/L.
Age Range Selection	Menu specified selection.	User designated option allows user to specify any designed age range from 1-7 years.
Multiple Runs	Multiple model runs are made manually; i.e., the user specifies input for each run, and saves output in Results Out file.	Allows user to input a range of lead values for one medium (air, diet, drinking water, dust or soil), and make a series of model runs in which lead levels in the specified medium are varied over the specified range.
X-axis Scaling	Set by program.	Allows user to set scaling of X-axis of probability density function and probability percent graphs manually.
Overlay Graph Files	Not available; graphs of individual model runs can be displayed and/or printed.	Allows user to save graph output (i.e., probability density function or probability percent graphs) from multiple runs to an file and print output of multiple runs on a single graph.
Text Files	Data from individual model runs can be saved to a file.	Allows user to save the data output of individual or multiple model runs, in user specified ASCII text files; these files can be reviewed and printed in Lead and/or imported into a word or processing program after exiting from Lead.
Blood vs. Media Concentration: Model Runs (Graph Output)	Not available.	Allows user to input a range of lead values for one primary medium (air, diet, drinking water, dust or soil), and produce a series of model runs in which lead levels in the specified medium are varied over the specified range. The output of each run is captured in an X-Y plot of mean blood lead vs. medium lead.
Blood vs. Media Concentration: Find	Not available.	Calculates a lead level in the user-specified medium that is associated with a specified mean blood lead.

TABLE B-3. SUMMARY OF REVISIONS TO LEAD UPTAKE BIOKINETIC MODEL SOFTWARE FROM LEAD 0.5 TO LEAD 0.99d

Parameter or Feature	Lead 0.5 (December, 1990)	Lead 0.99d (January, 1994)
Batch Mode Model Runs	Not available.	Accepts a properly formatted ASCII text file (*.DAT) containing data on lead exposure and blood lead levels for a sample population, and calculates predicted blood lead levels for each individual. The output is saved as a *.TXT file that can be viewed within Lead or imported into a text editor (e.g., word processor), and as a *.ASC file that can be imported into PBSTAT for selected statistical analysis and graphic display.
PBSTAT	Not available.	Accepts the output of batch mode operations (i.e., *.ASC files) and delivers statistical and graphic output selected by the user from a menu. PBSTAT can be accessed from the main menu of Lead, or externally with a DOS command (PBSTAT). The printer options in PBSTAT are the same as those in PBPLOT.
PBPLOT	Not available.	Produces graph prints from Lead. It is accessed with a DOS command (PBPLOT). PBPLOT performs the same functions as "Graphics Selection Menu" in Lead, however, it produces graph prints at much higher resolutions (e.g. HP Plotter and PostScript printer graphs). PBPLOT accepts *.LAY and *.PRM files produced with Lead.
Main Menu		Restructured.
Model Iteration Period	One month with no user option.	One day with user option.
"Fetal" Model	Maternal blood lead is calculated based on user-defined exposure data and a maternal biokinetic model, and newborn blood lead (BPbn) is calculated from a biokinetic model.	Not available.
Soil Intake	Default value is 100 mg/day for all ages.	Default values are: age (yr) / soil intake (mg/day) 0-1 / 85 1-2 / 135 2-3 / 135 3-4 / 135 4-5 / 100 5-6 / 90 6-7 / 85
View/Print Data File	User must exit Lead and import *.TXT data files into a text editor (e.g., word processor) to view or print data.	Allows the user to view and print *.TXT data files within Lead.

TABLE B-3 (cont'd). SUMMARY OF REVISIONS TO LEAD UPTAKE BIOKINETIC MODEL SOFTWARE FROM LEAD 0.5 TO LEAD 0.99d

Parameter or Feature	Lead 0.5 (December, 1990)	Lead 0.99d (January, 1994)
Contribution of soil lead to indoor dust lead	Default value function is 0.28 μg Pb/g dust per μg Pb/g of soil.	Default value function is 0.70 μg Pb/g dust per μg Pb/g of soil
Outdoor air lead concentration	Default value is 0.2 μg/m³.	Default value is 0.1 μg/m³
Dietary lead intake	Default values were based on a preliminary analysis of 1988 FDA survey data.	Default values are based on a more detailed analysis of FDA 1988-90 data
Editing of default biokinetic parameters	Coding error resulted in changes to RBCSUM time constants (e.g., TPLUR) not being accepted, as a result the UBM appeared to be insensitive to changes in TPLUR.	Coding error corrected.
Euler Algorithm	Forward Euler algorithm calculates the increase compartmental lead mass over the iteration interval as the total lead inflow to the compartment, minus the total lead outflow at the beginning of the interval.	Backward Euler algorithm calculates the increase compartmental lead mass over the iteration interval as the total lead inflow to the compartment, minus the total lead outflow at the end of the interval.
Tissue Pb and Excretory Transfer Coefficients		Revised
Non-linear GI Absorption Model	"Non-integrated" approach: Saturable absorption coefficients are calculated for each medium (soil, dust, drinking water, etc.) based on intakes from each medium, and are used to calculate media-specific uptakes which are summed to yield total uptakes.	"Integrated" approach: Intakes from all media are considered in the calculation of the saturable absorption coefficients for each medium (via SATURATION(i))

REGION 10

BUNKER HILL MINING AND METALLURGICAL COMPLEX, ID
August 30, 1991

SITE HISTORY/DESCRIPTION

The Bunker Hill Mining and Metallurgical Complex site is a 21-square-mile area centered around an inactive industrial mining and smelting site, and includes the Cities of Kellogg, Smelterville, Wardner, Pinehurst, and Page, in Shoshone County, Idaho. The inactive industrial complex includes the Bunker Hill mine and mill, a lead smelter, a zinc smelter, and a phosphoric acid fertilizer plant, all totaling several hundred acres. Furthermore, the site includes the South Fork of the Coeur d'Alene River, an alluvial floodplain bordered by mountains, numerous valleys and gulches, and vegetated residential areas. In 1886, the first mill for processing lead and silver ore was constructed at the site. Operations were expanded in later years with the addition of a lead smelter; a blast furnace; and electrolytic zinc, sulfuric acid, phosphoric acid, and fertilizer plants. Onsite operational and disposal practices have caused the deposition of hazardous substances (e.g., metals) throughout the valley via airborne particulate deposition, alluvial deposition of tailings dumped in the river, and migration from onsite sources. Initially, most of the solid and liquid residue from the complex was discharged into the river. When the river flooded, these materials were deposited onto the valley floor, and have leached into onsite soil and ground water. Although some of the industrial wastes have been removed and disposed of offsite, thousands of tons of sludge, tailings, flue dust, and other wastes still remain onsite. Contamination at the site is a result of tailings deposition in the floodplain, and airborne deposition from smelter and mill complex emissions. A fire in 1973 severely reduced air pollution control capacity at the lead smelter. A 1974 public health study and concurrent epidemiologic and environmental investigations concluded that atmospheric emissions of particulate lead from the active smelter were the primary sources of elevated blood lead levels in local children. In 1977, two tall stacks were added to disperse contaminants from the complex. The complex ceased smelter operations in 1981, but continued limited mining and milling operations from 1988 to early 1991. In 1989, EPA began a removal program to excavate lead-contaminated soil from affected residential properties. Federal and State agencies have designated a 21-square-mile study area, which has been divided into populated areas and non-populated areas for remediation. This ROD addresses contaminated residential soil within the populated areas of the site, and includes four incorporated communities and three unincorporated residential areas as operable unit 1. The nonpopulated areas of the site as well as all other contaminated media in the populated areas (e.g., house dust, and commercial properties) will be addressed in a future ROD. The primary contaminants of concern affecting residential area soil are metals including arsenic and lead.

SELECTED REMEDIAL ACTION

The selected remedial action for this site includes soil sampling; excavating contaminated soil and sod exceeding 1,000 mg/kg lead on approximately 1,800 residential properties, and replacing it with clean soil and sod; disposing of the contaminated soil and sod at an onsite repository; capping the repository; placing a visual marker if lead levels in soil exceed 1,000

mg/kg below the depth of excavation; revegetating the area; conducting long-term environmental monitoring; and implementing institutional controls including deed and land use restrictions. The estimated present worth cost for this remedial action is $40,600,000, which includes an annual O&M cost of $460,000 for 30 years.

PERFORMANCE STANDARDS OR GOALS

Residential soil with lead concentrations greater than 1,000 mg/kg will be excavated and replaced with clean material resulting in mean soil lead concentrations in residential areas of approximately 200 to 300 mg/kg.

INSTITUTIONAL CONTROLS

Deed, land use, and other administrative restrictions will be implemented onsite.

KEYWORDS

Arsenic; Capping; Carcinogenic Compounds; Clean Air Act; Clean Water Act; Direct Contact; Excavation; Filling; Floodplain; Institutional Controls; Lead; Metals; O&M; Onsite Containment; Onsite Disposal; Public Exposure; RCRA; Soil; State Standards/Regulations; Wetlands

SITE SUMMARY

Dates of Previous RODs: None
Lead: Fund
Contaminated Media: Soil
Major Contaminants: Metals
Category:
Source control - final action

DECLARATION FOR THE RECORD OF DECISION

SITE NAME

Bunker Hill Mining and Metallurgical Complex Site

Populated Areas

Residential Soils Operable Unit

LOCATION

Cities of Kellogg, Smelterville, Wardner, Pinehurst and other residential areas within the site Shoshone County, Idaho

STATEMENT OF BASIS AND PURPOSE

This decision document presents the remedial action selected by the U.S. Environmental Protection Agency and the Idaho Department of Health and Welfare for the Populated Areas Residential Soils Operable Unit at the Bunker Hill Mining and Metallurgical Complex Site in northern Idaho. The remedy was chosen in accordance with CERCLA, as amended by SARA, and, to the extent practicable, the National Contingency Plan. This decision is based on Residential Soils Administrative Record file for this site, and the index is attached.

ASSESSMENT OF THE SITE

Actual or threatened releases of hazardous substances from this site, if not addressed by implementing the response action selected in this Record of Decision (ROD), may present an imminent and substantial endangerment to public health, welfare, or the environment.

DESCRIPTION OF THE REMEDY

The Residential Soils Operable Unit is the first unit to be addressed at Bunker Hill. Exposure to lead in residential soils has been identified as the primary health risk to children and pregnant women within the Populated Areas of the site. Residential soils are not a "principal threat" at this site (as defined by U.S. EPA-see Glossary), although they represent a significant lead exposure pathway to the local population.

Exposure to interior house dust and consumption of locally grown garden produce have also been identified as significant contaminant exposure pathways to people. Contaminants of concern for garden produce include lead and cadmium.

Remediation of residential soils will break the direct contact exposure pathway between people and those soils. In addition, implementation of the selected remedy will remove a source of metal-contaminated dust to home interiors (residential soils are a source of house dust), and provide safe garden areas.

The residential soils remedy consists of the following:

- o Removal of contaminated surficial soil

- o Placement of a visual marker if lead in soil concentrations exceed 1,000 ppm below the depth of excavation

- o Replacement with clean soil (these soils will function as a barrier between residents and underlying contaminated material)

- o Revegetation of yards

- o Disposal of contaminated materials

- o Dust suppersion during remediation

- o Institutional controls for barrier management

- o Long-term environmental monitoring for evaluation of remedial effectiveness

A Remedial Action Objective is to decrease the concentration of lead such that 95 percent or more of the children in the area have blood lead levels below 10 ug/dl. This remedial action is expected to achieve community mean soil lead concentrations of approximately 200 to 300 ppm by removal of soils exceeding the threshold level of 1,000 ppm lead. Approximately 1,800 residential properties will be remediated based on this criterion. U.S. EPA and IDHW have determined that residential yards cleaned up in 1989, 1990, and 1991 were done so in a manner consistent with this Record of Decision. These properties will be included in the Institutional Controls Program.

To meet the health based Remedial Action Objectives, contaminated fugitive dust must be controlled and lead concentrations in home interior dust must be reduced. It is expected that there will be at least one other Record of Decision that will address fugitive dust, interior dust, and all other remaining issues for the site.

STATUTORY DETERMINATIONS

The selected remedy is protective of human health and the environment, complies with federal and state requirements that are legally applicable or relevant and appropriate to the remedial action, and is cost-effective. This remedy utilizes permanent solutions and alternative treatment technologies to the maximum extent practicable. However, because treatment of the metal-contaminated residential soils was found to be not practicable, this remedy does not satisfy the statutory preference for treatment as a principal element of the remedy. Treatment was determined to be impracticable based upon effectiveness and cost factors.

Because this remedy will result in hazardous substances remaining onsite above health-based levels, a review will be conducted within 5 years after commencement of remedial action to ensure that the remedy continues to provide adequate protection of human health and the environment.

Richard P. Donovan

Director

Idaho Department of Health and Welfare

DATE August 26, 1991

Dana A. Rasmussen

Regional Administrator

U.S. EPA Region 10

DATE August 30, 1991

RECORD OF DECISION SUMMARY

Site Name: Bunker Hill Mining and Metallurgical Complex Site

Populated Areas

Residential Soils Operable Unit

Location: Cities of Kellogg, Smelterville, Wardner, Pinehurst; and other residential areas within site boundaries

Shoshone County, Idaho

1 SITE DESCRIPTION

The Bunker Hill Mining and Metallurgical Complex Superfund Site is located in Shoshone Count, in northern Idaho, at 47 degrees 5' north latitude and 116 degrees 10' west longitude (Figure 1-1). The site lies in the Silver Valley of the South Fork of the Coeur d'Alene River (SFCDR). The Silver Valley is a steep mountain valley that trends from east to west. Interstate Highway 90 crosses through the valley, approximately parallel to the SFCDR. The site includes the town of Pinehurst on the west and the town of Kellogg on the east (Figure 1-2) and is centered on the Bunker Hill industrial complex. The site has been impacted by over 100 years of mining and 65 years of smelting activity. The complex occupies several hundred acres in the center of the site between the towns of Kellogg and Smelterville.

Populated and Non-populated Areas of the Site
(See Figure 1-2 in Original Document)

The agencies (U.S. Environmental Protection Agency (U.S. EPA) and Idaho

Department of Health and Welfare (IDHW)) have designated a 21-square-mile study area for purposes of conducting the Remedial Investigation/Feasibility Study (RI/FS), which has been divided into Populated Areas and Non-populated Areas. This Record of Decision (ROD) addresses contaminated residential soils within the Populated Areas of the site. Soils throughout the site have been contaminated by heavy metals to varying degrees, through a combination of airborne particulate deposition, alluvial deposition of tailings dumped into the river by mining activity, and contaminant migration from onsite sources. Onsite sources include the industrial complex, tailings and other waste piles, barren hillsides, and fugitive dust source areas located throughout the site.

The Populated Areas of the site consist of four incorporated communities and three unincorporated residential areas. Except for the eastern portion of Kellogg, all of these communities lie south of U.S. Interstate 90 (I-90), between the highway and steep hillsides to the south. Portions of the residential areas lie within the floodplain of the South Fork of the Coeur d'Alene River.

This ROD addresses currently established residential areas. The city of Kellogg (see Figure 1-3) is 6 miles east of the western edge of the site and approximately 1 mile east of the smelter complex. The population is estimated to be 2,600 with about 1,100 residences. The next largest population center is the city of Pinehurst (see Figure 1-4) with 700 residences and about 1,700 people. It is located on the western edge of the site, about 1 mile south of I-90. Smelterville (see Figure 1-5), with a population of about 450 and 270 residences, is approximately 3 miles east of the western edge of the site and lies along a minor arterial road linking it to Pinehurst and Kellogg. The town is about 1 mile west of the smelter complex. The city of Wardner (see Figure 1-6) is contiguous with the southeast portion of Kellogg and is approximately 6 miles east of the western boundary of the site. The population of Wardner is currently about 300 people with 130 residences. The unincorporated community of Page (see Figure 1-7) is about 1 mile east of the western edge of the site. Most of the land is owned by American Smelting and Refining Company (ASARCO), while the homes are owned by the residents. Population of Page is estimated to be about 100 to 150 people, and the area includes 65 residences. Two unincorporated residential areas located along the eastern site boundary are Elizabeth Park and Ross Ranch with populations estimated to be 120 and 50 people, respectively.

2 SITE HISTORY AND ENFORCEMENT ACTIVITIES

2.1 SITE HISTORY

The Bunker Hill Superfund Site is part of the Coeur d'Alene Mining District located in northern Idaho and western Montana. Gold was first discovered in the district in 1883. The first mill for processing lead and silver ores at the Bunker Hill site was constructed in 1886 and had a capacity of 100 tons of raw ore per day. Other mills subsequently were built at the site and the milling capacity ultimately reached 2,500 tons per day.

The Kellogg-based Bunker Hill and Sullivan Mining Company, incorporated in 1887, was the original owner and operator of the Bunker Hill complex. In 1956, the name was

changed to the Bunker Hill Company and in 1968, Gulf Resources and Chemical Company of Houston, Texas, purchased the company and operated the smelter until it was closed is late 1981. The complex was purchased in 1982 by the Bunker Limited Partnership (BLP), headquartered in Kellogg, Idaho. BLP subsequently sold portions of the complex properties to several related or affiliated entities including:

- o Syrina Minerals Corporation
- o Crescent Mine
- o Bunker Hill Mining Company (U.S.), Inc.
- o Minerals Corporation of Idaho

The Bunker Mining Company resumed mining and milling operations in 1988 and subsequently ceased those operations in 1991.

The Bunker Hill and Sullivan Mining Company was originally involved only in mining and milling lead and silver ores from local mines. From 1886 until 1917, the lead and silver concentrates produced at the site were shipped to offsite smelters for processing. Construction of the lead smelter began in 1916 and the first blast furnace went online in 1917. Over the years, the smelter was expanded and modified. At the time of its closure in 1981, the lead smelter had a capacity of over 300 tons of metallic lead per day. An electrolytic zinc plant was put into production at the site in 1928. Two sulfuric acid plants were added to the zinc facilities in 1954 and 1966, and one sulfuric acid plant was added to the lead complex in 1970. When it was closed in 1981, the zinc plant's capacity was approximately 285 tons per day of cast zinc. A phosphoric acid plant was constructed at the site in 1960 and a fertilizer plant was built in 1965. The primary products from these plants were phosphoric acid and pellet-type fertilizers of varying mixtures of nitrogen and phosphorus. The industrial complex ceased operation in 1981 except for limited mining and milling operations mentioned above.

Control of atmospheric emissions, solid waste disposal, and wastewater treatment at the Bunker Hill complex evolved with changing technologies and regulations. Initially, most liquid and solid residue from the complex was discharged into the South Fork of the Coeur d'Alene River and its tributaries. The river periodically flooded and deposited waste material laden with lead, zinc, and other heavy metals onto the valley floor. Operation and disposal practices caused deposition of hazardous substances throughout the valley. Leaching of these deposits through the soil has contributed to heavy metal contamination of the river and groundwater.

A 1973 fire in the baghouse at the lead smelter main stack severely reduced air pollution control capacity. Total particulate emissions of about 15 to 160 tons per month, containing 50 to 70 percent lead, were reported from the time of the fire through November 1974. This compares to emissions of about 10 to 20 tons per month prior to the fire. The immediate effects of increased total lead emissions and high total lead in air content were observed in a 1974 public health study where a significant number of children had elevated blood

lead levels. Lead smelter stack emissions following the 1973 baghouse fire are a significant source of current site contamination.

In 1977, tall stacks (>600 feet) were added at both the zinc and lead smelters to more effectively disperse contaminants from the complex. These devices decreased sulfur oxides concentrations in the late 1970s. The smelter and other Bunker Hill Company activities ceased operation in December 1981, and portions of the smelter complex have since been salvaged for various materials, machinery, and scrap.

Although in recent years some wastes have been shipped offsite for disposal in landfills, thousands of tons of sludges, tailings, flue dust, and other wastes remain at the complex. These materials contain high levels of arsenic, lead, and other metals.

2.2 INITIAL INVESTIGATIONS

Contaminated air, soils, and dusts have been identified as contributors to elevated blood lead levels in children living in the Populated Areas of Bunker Hill site. Environmental media concentrations of site contaminants of concern in the Populated Areas are strongly dependent on distance from the smelter facility and industrial complex. Residential areas nearest the smelter complex have shown the greatest air, soil, and dust lead concentrations; the highest childhood blood lead levels; and the greatest incidence of excess absorption in each of the studies conducted in the last decade.

Health effects of environmental contamination were first documented following the smelter baghouse fire and associated smelter emissions in 1973 and 1974. Up to 75 percent of the preschool children tested within several miles of the complex had blood lead levels at that time that exceeded Centers for Disease Control (CDC) criteria. Several local children were diagnosed with clinical lead poisoning and required hospitalization. Lead health surveys conducted throughout the 1970s confirmed that excess blood lead absorption was endemic to this community. Concurrent epidemiologic and environmental investigations concluded that atmospheric emissions of particulate lead from the active smelter were the primary sources of environmental lead that affected children's blood lead levels prior to 1981. Contaminated soils were also found to be a significant, however secondary, source of lead to children in the 1970s.

Following lead poisoning incidents in 1973, a number of activities were instituted to decrease lead exposures and uptakes in the community. In an August 1974 survey, 99 percent of the 1to 9-year-old children living within 1 mile of the smelter were found to have blood lead levels in excess of 40 ug/dl. The frequency of abnormal lead absorption (defined at the time as greater than or equal to 40 ug/dl was found to decrease with increasing distance from the smelter. Following the announcement of these results, emergency measures were initiated to reduce the risk of lead intoxication. These measures included: chelation of children with blood lead over 80 ug/dl, purchase and destruction of as many homes as possible within 0.5 mile of the smelter, distribution of "clean" soil and gravel to cover highly contaminated areas, initiation of a hygiene program in the schools, and reduction of ambient air lead levels through reduction of smelter emissions. Street cleaning and watering in dust-producing areas occurred during several periods in the late 1970s. Subsidies were provided by the Bunker Hill Company to

residents for the purchase of clean top soil, sand, gravel, grass seed and water, thereby promoting some yard cover in the community.

An analysis of historical exposures to children who were 2 years old in 1973 suggests a high risk to normal childhood development and metal accumulation in bones because of extreme exposures; these exposures could offer a continuing lead body burden in these children because of its long physiologic half life. Females who were 2 years of age during 1973 are now of childbearing age and, even with maximum reduction in current exposure to lead, the fetus may be at risk because of resorption of bone lead stores in the young women.

Following smelter closure in late 1981, airborne lead contamination decreased by a factor of 10, from approximately 5 ug/m3 to 0.5 ug/m3. A 1983 survey of children's blood lead levels demonstrated a significant decrease in community exposures to lead contamination; however, the survey also found that several children, including some born since 1981, continued to exhibit blood lead levels in excess of recommended public health criteria. Accompanying epidemiological analyses suggested that contaminated soils and dusts represented the most accessible sources of environmental lead in the community.

Childhood mean blood lead levels have continued to decrease since 1983. These decreases are likely related to a nationwide reduction in dietary lead; reduced soil, dust, and air levels in the community; intake reductions achieved through denying access to sources; and the increase in family and personal hygiene practiced in the community. The latter is reflected in the implementation of a comprehensive Community Health Intervention Program in 1984 that encourages improved hygienic (housekeeping) practices, increased vigilance, parental awareness, and special consultation on individual source control practices such as lawn care, removals, and restrictions. The Community Health Intervention Program was initiated specifically to reduce the potential for excess absorptions and minimize total absorption in the population until initiation of remedial activities. Total blood lead absorption among the community's children has been reduced nearly 50 percent since 1983. The incidence of lead toxicity (blood lead > 25 ug/dl) has fallen from 25 percent to less than 5 percent for children in the highest exposure areas. Recent blood lead monitoring has shown 37 to 56 percent of area children surveyed exceeded the blood lead level of 10 ug/dl.

2.3 REMEDIAL INVESTIGATION/FEASIBILITY STUDY (RI/FS)

The Bunker Hill site was placed on the National Priorities List (NPL) in September 1983 (48 FR 40658). RI/FS activities were initiated in late 1984 following completion of the 1983 Lead Health Study.

The Bunker Hill Site Characterization Report (SCR) was the first step in the RI process. The objective of the SCR was to describe and analyze existing information. The existing information included files from federal, state, and local agencies, as well as information obtained from past and present owners and operators of the industrial complex. The SCR was then used to identify data gaps and develop work plans for the remedial investigation.

In recognition of the history and complexity of this site, and the continuing need

for active health intervention efforts, the EPA and IDHW developed an integrated project structure for RI/FS activities. The site was divided into two principal portions--the Populated Areas and the Non-populated Areas. The Populated Areas include several cities, all residential and commercial properties located within those cities, and other residential properties. The Non-populated Areas include the smelter complex, river floodplain, barren hillsides, groundwater, air pollution, and industrial waste components of the site.

While separate RI/FS efforts were initiated for each portion of the site, U.S. EPA Region 10 retained oversight and risk assessment responsibilities for both. IDHW conducted the Populated Areas RI/FS. The Non-populated Areas RI/FS is being conducted by Gulf Resources & Chemical Corporation under a U.S. EPA Administrative Order on Consent signed by U.S. EPA in May 1987. Table 2-1 lists the major geographic features and investigation emphases.

Table 2-1 Major Features and Investigation Emphasis
(See Data in Original Document)

2.4 HISTORY OF CERCLA ENFORCEMENT

Several companies have been identified by U.S. EPA as potentially responsible parties (PRPs) for the Bunker Hill Superfund Site. Table 2-2 lists the PRPs for Bunker Hill and the dates they were notified. The PRPs represent a combination of past and present property owners, owners and operators of the various smelting, processing, and production facilities located within the industrial complex, and upstream mining companies that were responsible for tailings discharges into the South Fork of the Coeur d'Alene River that have contributed to the contamination of the site.

Table 2-2 Potentially Responsible Parties Identified for the Bunker Hill Superfund Site
(See Data in Original Document)

In 1989, U.S. EPA recovered $1.4 million from Gulf Resources & Chemical Corporation in a settlement regarding Superfund money spent during the removal action in 1986. Agency oversight costs associated with the Non-populated RI/FS have been received from Gulf Resources & Chemical Corporation for 1987 through 1989. On May 2, 1990, U.S. EPA filed a civil action for penalties against Bunker Limited Partnership for failure to respond to U.S. EPA's October 1988 request for information. The case is still pending in U.S. District Court in Boise, Idaho.

2.5 REMOVAL ACTIONS

There have been two Superfund-financed removal actions (1986 and 1989 residential soils); one removal action was financed by the PRPs but performed by the agencies (1990 residential soils); and there have been three PRP-performed removal action (1989 Smelter Complex Stabilization, 1990 hillsides revegetation, and 1991 residential soils, etc.).

In 1986, 16 public properties (parks, playgrounds, and road shoulders) were selected for an immediate removal action because these properties contained high concentrations of lead and were frequented by many area children. The action consisted of placing a barrier between children and the underlying contaminated soil. Six inches of contaminated materials were excavated, and clean soil, sod and/or gravel were imported for replacement. Excavated material was temporarily stored within site boundaries at property owned by the Idaho Transportation Department (ITD).

In 1989, the U.S. EPA and IDHW began a residential soil removal program. The program prioritized yards that had a lead concentration greater than or equal to 1,000 ppm and housed either a young child or a pregnant woman. This action consisted of removing 6 to 12 inches of contaminated material from yards and replacing it in kind with clean material. Contaminated soils were again stored at the ITD property within site boundaries. In 1989, yard soil replacement was completed at 81 homes and 2 apartment complexes within the Populated Areas of the site.

An Administrative Unilateral Order was issued October 24, 1989 (U.S. EPA Docket Number 1989-10-21-106), to Bunker Limited Partnership, Minerals Corporation of Idaho, Bunker Hill Mining Company, (U.S.) Inc., and Gulf Resources and Chemical Corporation. The purpose of the order was to implement actions to stabilize several problem areas within the industrial complex. Actions required by the order included immediate cessation of salvaging activities onsite, establishment of site access restrictions, development of a dust control plan, and stabilization and containment of the copper dross flue dust pile.

An Administrative Unilateral Order was issued to all named PRPs on May 15, 1990 (U.S. EPA Docket No. 1090-05-25-106(a)), which required the continuation of the residential soil removal program within the boundaries of the Superfund site. Settlement of this order resulted in an agreement between U.S. EPA and eight of the PRPs (Gulf Resources & Chemical Corporation, Hecla Mining Company, ASARCO, Inc., Stauffer Chemical Company, Callahan Mining Corporation, Coeur d'Alene Mines Corporation, Sunshine Precious Metals, Inc., and Union Pacific Railroad) for payment of $3,180,000 to U.S. EPA (U.S. EPA Docket Number 1090-05-35-106) for performance of the 1990 residential soil removal action. Yard soil removal and replacement for an additional 130 yards were performed in 1990. Excavated soils from this removal action were stored at the Page Ponds tailing impoundment.

An Administrative Order on Consent to implement hillside stabilization and revegation work was entered into between U.S. EPA and Gulf Resources & Chemical Corporation, and Hecla Mining Company, on October 1, 1990 (U.S. EPA Docket No. 1090-10-01-106). The objectives of this Order are to control erosion by reestablishing a native, closed, coniferous forest and understory vegetative cover to approximately 3,200 acres of barren hillsides and to perform terrace repair and construction of detention basins, and repair of the rockslide areas in Wardner and Smelterville. Planting of trees is scheduled to be completed in 1996.

In July of 1991, an Administrative Order on Consent (U.S. EPA Docket No. 1091-06-17-106(a)) was entered into between U.S. EPA and nine PRPs (Gulf Resources &

Chemical Corporation, Hecla Mining Company, ASARCO, Inc., Stauffer Chemical Company, Callahan Mining Corporation, Coeur d'Alene Mines Corporation, Sunshine Precious Metals, Inc., Union Pacific Railroad, and Sunshine Mining Company) that required the PRPs to perform the residential soil removal program. It is expected that approximately 80 more properties will be cleaned up this year. As in 1990, excavated soils were stored at the Page Ponds tailing impoundment. Under this Order, the parties have also agreed to undertake sitewide dust control actions; monitor air, groundwater and surface water; enhance the fire fighting capability at the industrial complex; and provide funding to purchase high-efficiency vacuums for loan as part of the Health Intervention Program.

3 HIGHLIGHTS OF COMMUNITY PARTICIPATION

There has been a long history of community relations activities in the Silver Valley. Since discovery of elevated blood leads in children in 1974, the IDHW, Panhandle Health District (PHD), and the CDC have continually worked with area residents to reduce exposures to lead. In 1985 the Shoshone County Commissioners selected a nine-member Task Force to serve as a liaison between the Bunker Hill Superfund Project Team (comprised of representative of U.S. EPA and IDHW and contractors) and the community. The PHD was contrasted by IDHW to perform community relations tasks for the Bunker Hill Superfund Site. A full-time IDHW staff person has also been stationed onsite from mid-1987 to present. Part of their duties is to assist in community relating activities when needed.

The focus of community contact has been the nine-member Silver Valley Task Force. There have been 35 public task force meetings since May of 1985. These meeting consisted of presentations by the Bunker Hill Project Team with time for questions and statements from both the Task Force and the general community. Twenty-three fact sheets have been produced since May 1985 to discuss various aspects of the RI/FS activities at the site. Site records have also been made available to the public through four public information repositories. The community was involved in the selection of activities associated with residential soil removal actions through a public comment period. This experience, along with the opportunity to observe the cleanup activity over the last 2 years, has helped familiarize the community with the remediation of residential soils.

A series of meetings has been held between the PHD and local planning and zoning commissions, city councils, and county commissioners to help develop the "Evaluation of Institutional Controls for the Bunker Hill Superfund Site." Institutional control development presentations were also made to local business and community groups.

The "Risk Assessment Data Evaluation Report," the "Residential Soils Focused Feasibility Study," the "Proposed Plan for Cleanup of Residential Soil within the Population Areas of the Bunker Hill Superfund Site," and "An Evaluation of Institutional Controls for the Bunker Hill Superfund Site" were released for public review April 29, 1991. These four documents were made available in the administrative record file, which is located at the Kellogg City Hall, and the four information repositories, which are located at the Kellogg City Hall, Kellogg Public Library, Smeltsville City Hall, and Pinehurst/Kingston Library. The notice of availability of the documents was published in the "Shoshone News Press" from April 26,

through April 30, 1991. The notice outlined the remedial alternatives evaluated and identified the proposed alternative. A public comment period was established for April 29 to May 31 and was extended to June 30, 1991, after a request to extend the period was received. Extension of the public comment period was published in the "Shoshone News Press" May 24 through 26, 1991. A public hearing was held May 23, 1991, to answer questions and take comments. There were approximately 100 attendees at the meeting. A transcript of questions asked and answers given at the public hearing is included in the Administrative Record. Responses to written comments are included in the Responsiveness Summary, which is part of this Record of Decision.

4 SCOPE AND ROLE OF OPERABLE UNIT

The rationale for separating the Bunker Hill RI/FS into two parts involved both data availability and confidentiality issues associated with an investigation of private residential properties within the Populated Areas. With both environmental data and an abundance of human health related data, collected as part of the epidemiological studies, the agencies believed that the Populated Areas RI/FS could best be completed by government agencies in order to honor confidentiality agreements with individuals and individual property owners.

The RI--Risk Assessment Data Evaluation Report (RADER) for the Populated Areas of the Site--has been completed. The residential soils feasibility study is also complete and is the first unit to be addressed in a Record of Decision. The other units that are related to the Populated Areas investigation that have not been addressed in a decision document include: house dust, commercial properties, and road shoulders and rights-of-way. The agencies originally expected to address these issues in a second ROD in 1992; however, the PRPs have approached U.S. EPA and IDHW with a proposal for a sitewide cleanup that involves all facets of both the Populated and Non-populated Areas. The effort to complete the Residential Soils ROD was maintained, because soils are a primary risk to the residents; however, consolidation of all (see Table 2-1) remaining issues into what is referred to as the expedited FS is ongoing. The expedited FS is expected to support a second ROD for the site that will address all contaminated areas and media not covered in this ROD.

The RADER concluded that subchronic lead absorption among young children is the most significant health risk posed by this site. The greatest risks to young children are associated with ingestion of residential yards soils, house dusts, and locally grown produce. Exposure to residential soils is a primary health risk to area residents, although residential soils are not a "principal threat" as defined by U.S. EPA. The remedial action described in this ROD is intended to minimize direct contact with and ingestion of lead-contaminated residential soils by excavation and replacement of those soils with clean material. While yard soils represent a primary risk to local residents, it is important to recognize that yard soils represent only one component of exposure in these communities. Other sources of contamination within the site must be addressed to prevent additional population exposures and recontamination of residential soils because of contaminant migration. No direct action is being taken for house dust lead reduction at this time; however, it is expected that house dust concentrations will decrease as yard soil lead concentrations decrease and fugitive dust sources are controlled. Part of the ongoing Health Intervention Program will be to lend high-efficiency home vacuum cleaners to

interested residents. Fugitive dust control efforts undertaken as part of the 1991 removal action will further reduce exposures and the transport of contaminated materials.

Use of a threshold level of 1,000 ppm lead (i.e., remedial action at any yard with a lead concentration of 1,000 ppm or above) will result in residential community mean soil lead concentrations of approximately 200 to 300 ppm. Current community mean soil lead concentrations are approximately 3,000 ppm. The goal is to reduce soil lead concentrations such that mean blood lead levels are below 10 ug/dl and the risk for any individual child to have a blood lead level that exceeds 10 ug/dl is minimized.

Locally grown produce is a potentially significant exposure route for cadmium and lead to pregnant women as well as young children. This action will provide for safe produce gardening areas to ensure that this exposure pathway is minimized. Currently, the Health Intervention Program recommends that produce grown in local gardens not be consumed.

There are approximately 2,700 residential properties onsite. Of those, approximately 50 percent have been sampled. Of the yards sampled, 65 percent have surface soil concentrations of lead greater than or equal to 1,000 ppm. If the unsampled yards show a similar distribution, this action is expected to involve remediation of 65 percent (approximately 1,800) of the residential yards within the site.

5 SITE CHARACTERISTICS

5.1 PHYSICAL SETTING

Topography of the Silver Valley consists of an alluvial floodplain bordered on the north and south by steep mountains. The floodplain ranges in width from about 0.1 mile east of Kellogg to approximately 0.9 mile near Smelterville. The elevation of the valley floor ranges from 2,160 feet above mean sea level at the west end to 2,320 feet at the east end of the project site. The valley floor is nearly level, with slopes typically less than 1 percent. The mountains rising from the valley range from 500 to 2,500 feet above the valley floor. The mountainsides typically exhibit slopes of 45 to 90 percent and at some points exceed 110 percent. Numerous valleys and gulches cut through the mountains and generally trend north to south, intercepting the valley of the South Fork Coeur d'Alene River.

Most residences are located on the valley floor or at the toe of the hillside slopes. Valley floor soils were formed from alluvially deposited materials and have been strongly influenced by mine tailings placed in the river as a result of past mining activity. In general, the alluvial valley-fill deposits are comprised of silty to clayey sand and gravel. Soil parent materials at the toe of the steep slopes are colluvial and mixed colluvial/alluvial and are highly erosive. Residential soils have been modified by typical excavation and backfill practices utilized during home construction.

Vegetation in the residential areas includes conifer and deciduous trees, grass lawns varying in quality with level of maintenance, some vegetable and flower gardens, and native grasses in undeveloped or steeply sloping areas.

The meteorology of the site is dominated by mountain/valley drainage winds related to the local topography. The orientation of the valley effectively channels winds in an east-west direction. Nocturnal winds average 4.5 mph and tend to be from the east. Late morning and afternoon winds are from the west and southwest, averaging approximately 8 mph. The mean precipitation of the area ranges from 30.4 inches at Kellogg to 40.5 inches at the nearby city of Wallace, 10 mile east (upstream) of the site. Data from the National Weather Service collected from 1951 to 1980 show an annual mean temperature in Kellogg of 47.2 degrees F. A record high of 111 degrees F was reached on August 5, 1961, and a record low of -36 degrees F on December 30, 1968. On the average, 28 days per year reach a high temperature of 90 degrees F or greater, and 143 days reach a low reach a low of 32 degrees F or less.

5.2 NATURE AND EXTENT OF CONTAMINATION

The scope of the Populated Areas RI included residential soil, fugitive dust source, house dust, and air monitoring studies. Contaminants of concern for residential soils are antimony, arsenic, cadmium, copper, lead, mercury, and zinc. Lead has been identified as the primary contaminant of concern based on health studies.

Residential yard soil concentrations are presented in Table 5-1. The right-hand column of the table presents background mean concentrations for comparison. Data from the residential yards show that metal concentrations in surficial soils are greatly increased over background. Residential soil contamination concentrations decrease with increasing distance from the mill and smelter complex and result from a variety of historical industrial activities.
If the columns or type in the table(s) below appear misaligned, please press Ctrl+V and scroll right to view. When done, press ESC to restore your original view.

TABLE 5-1
SUMMARY OF RESIDENTIAL SOIL METAL CONTAMINATION LEVELS - PINEHURST

	Concentration, ppm, dry wt. (ppm)							
	Arith	Geom.						Back ground
Metal	Mean	Median	Mean	95%ile	Min.	Max.	N	Mean
As	30	21	23	73	7	123	100	<10
Cd	6	6	5	13	1	37	100	0.8
Cu	43	40	39	85	17	167	100	28
Hg	0.5	0.4	0.4	1	0.1	4	100	0.1
Pb	683	501	463	1260	63	7990	100	43
Sb	9	7	8	19	5	41	100	1
Zn	474	394	389	1060	99	2300	100	95

Metal contamination to depths as great as 3 feet have been identified in residential soils. Contamination sources at this depth are primarily alluvially deposited tailings.

Table 5-2 summarizes the percentage and number of properties within each community with yard soil lead concentrations above 1,000 ppm.

Table 5-2 Residential Properties With Lead Concentrations Above 1,000 ppm Lead
(See Data in Original Document)

Soil samples collected from 40 different yards were analyzed for other potential contaminants such as extractable organic compounds, chlorinated pesticides, PCBs, and mercury. Most organic analytes were not detected. However, occasional detections were noted for phthalate esters (plasticizer compounds), some polynuclear aromatic hydrocarbons (i.e., naphthalene, phenanthrene, fluoranthene, pyrene, benzo(b) fluoranthene, and benzo(a)pyrene as constituents of fossil fuels and their combustion products), and polychlorinated bipehnyls (PCBs as components of electrical transformer dielectric fluids). Chlorinated pesticides were detected in several samples in each town. For those pesticides observed, the frequencies of detection range from a low of 14 percent for aldrin, lindane, and heptachlor to a high of 100 percent for DDT isomers and metabolites, chlordane, and heptachlor epoxide. Greatest concentrations and frequencies of detection for pesticides in soils were found in Smelterville, Kellogg, and Wardner, with significantly lower levels in Page. Presence of organic and pesticide contaminants in residential soil could not be related to mining and industrial activities with the site.

Many residential streets and roads do not have paved curbs and sidewalks. Metals concentrations from samples collected from the surface inch of the road shoulders are shown in Table 5-3. Metals concentrations in roadside samples show considerable variation, both geographically and within towns. Samples from Smelterville ranged from 249 to 60,100 ppm Pb; 3 to 487 ppm Cd; and 19 to 810 ppm As. Samples from the Sunnyside area of Kellogg (north of I-90) averaged 1,935 ppm Pb; 19 ppm Cd; and 71 ppm As. Old Town area (south of I-90) samples averaged 4,497 ppm Pb; 28.6 ppm Cd; and 81 ppm As. Wardner and Pinehurst area samples were notably lower, averaging 1,385 ppm Pg; 15 ppm Cd; and 73 ppm As. Samples of street sweeper dust showed lead contents from 1,560 to 2,230 ppm and zinc levels exceeding 10,000 ppm (1 percent).

Table 5-3 Summary of Road Shoulder and Railroad Right-of-Way
(See Data in Original Document)

In 1988 and 1989, efforts were undertaken to assess recontamination at sites cleaned up in the summer of 1986. Removal actions implemented during 1986 included a 6-inch removal of contaminated soils and replacement with clean materials and sod in parks and playgrounds, and asphalting or gravel cover of roadsides and parking lots. Table 5-4 summarizes the original (preremediation) lead concentrations, remedial material (clean fill) lead concentrations, and the two recontamination assessment efforts.

Table 5-4 1986 "Fast-Track" Removal Efforts and Lead Recontamination Surveys
(See Data in Original Document)

The few sod samples collected suggest surface recontamination rates of 10 to 100 ppm/yr lead. No recontamination was evident in either the top inch or middle of the soil fill on sodded sites or play fields. Some recontamination was evident at the interface or replaced soils and top of the original cut. Whether this was due to contaminant migration, mixing at the time of placement, or imprecise layering of the sample is unknown. Rudimentary modeling has indicated that upward migration potential exists only in isolated areas where there is shallow groundwater.

Graveled areas, particularly those used as parking lots, showed significant recontamination. Because of the low rates of surface deposition, these increases likely resulted from the continual working of the original soil layers below the replacement materials or tracking of contaminants onto the site by vehicles.

Migration and transport of contaminated solids from the industrial complex and other fugitive dust sources are a major concern in both the Populated and Non-populated Areas of the site. Windblown dusts are potentially significant contributors to contaminant concentrations in human receptor media in the Populated Areas and have been identified as a major source of public complaint. Many of the identified fugitive dust sources are barren soils and impounded wastes and storage piles that can result in significant amounts of reentrained dusts.

Eighteen major barren areas identified as having a potentially significant impact on the residential areas were sampled during remedial investigations in 1986. Table 5-5 identifies the areas sampled, the respective size of each area, the number of samples collected, summary statistics for lead content in the minus 200mesh portion of the sample, and the average percentage (by weight) that passed the 200-mesh sieve. Antimony, arsenic, cadmium, copper, and zinc were also detected in all samples collected. Locations of the fugitive dust source areas samples are provided in Figure 5-1.

Table 5-5 Fugitive Dust Source Areas
(See Data in Original Document)

Potential Source Identification Map for Windblown Dusts
(See Figure 5-1 in Original Document)

Highest metal concentrations among fugitive dust sources were found adjacent to the concentrator building, with the lead concentration averaging about 230,000 ppm (23 percent), and arsenic and cadmium levels each at approximately 10,000 ppm (1 percent). Dust content for this sample was high with 30 percent of the solids passing 200-mesh sieve. The surrounding areas (11 and 12) also have relatively high metal contaminant levels that may be related to emissions from the concentrator area. Barren areas near Shoshone Apartments (Areas 11) and the Water Treatment Plant (Area 12) exhibit approximately 49,000 ppm (4.9 percent) and 43,000 ppm (4.3 percent) lead in surface dust, respectively. The arithmetic mean lead concentration for all fugitive dust source areas is 28,400 ppm (2.8 percent). Source areas near the smelter complex

and throughout the river floodplain routinely exhibited levels in excess of 2 percent lead. Percent of sample solids to pass the 200-mess sieve ranged from 6 to 68 percent, averaging 30 percent for all samples.

Air monitoring was used to investigate air contaminant transport mechanisms. Air monitor locations are shown in Figure 5-2. Total Suspended Particulate (TSP) data are summarized in Table 56. Metal content of filters collected on high dust event days (defined as days with TSP > 150 ug/m3) is summarized in Table 57. The 19 days in 1987 where blowing dust events were measured account for 43 percent of the Total Suspended Particulates (TSP) loading for the entire 116-day sampling season. The single highest day (September 2, 1987) alone accounted for nearly 10 percent of the total monitoring season loading. In 1989, the peak 10 days accounted for 48 percent of the loading for the 90day monitoring period.

Air Monitoring Locations
(See Figure 5-2 in Original Document)

Table 5-6 1987 and 1989 Air Monitoring Data TSP Data (ug/m-3)
(See Data in Original Document)

Table 5-7 Summary of Air Filter Metals Data (ug/m-3)--1987 and 1989 Event Monitoring
(See Data in Original Document)

Metal contaminant levels in house dusts are presented in Table 58. House dust metal contamination, and especially lead contamination, has decreased markedly since 1974. For example, the mean house dust lead concentration in Smelterville for 1974 was approximately 12,000 ppm (1.2 percent) and has decreased to a mean level in 1988 that is one-tenth the 1974 value (1,200 ppm). Prior to 1981, during smelter operations, the primary route for house dust lead contamination was airborne deposition of smelter lead particulate matter. Since 1981, house dust metals levels have been related to residential soil concentrations. Contaminated dust reach homes via deposition of windblown dusts or mechanical translocation of contaminated residential soils. Several studies indicated house dust lead levels in urban and smelter communities (exclusive of those impacted by interior leaded paints) are depended on lead levels in residential soils.

Table 5-8 Geometric Mean and Extreme House Dust Metal Concentrations 1974, 1975, 1983, and 1988 Lead Health Survey (ppm)
(See Data in Original Document)

5.3 CONTAMINANT MIGRATION

Soils within the site have been contaminated by heavy metals, to varying degrees, through a combination of airborne particulate deposition, alluvial deposition of tailings dumped

into the river by mining activities, and contaminant migration from onsite sources. Onsite sources include the smelter facility, industrial complex, tailings and other waste piles, barren hillsides, and other fugitive dust source areas located throughout the site. Since shutdown of the smelter, contaminant migration pathways of primary concern are fugitive dust, flooding that redeposits tailings into residential areas, water erosion that results in contaminated soil movement off of the hillsides, and human activities that either exacerbate the previous pathways or directly contaminate residential soils.

The current primary contaminant mechanism is airborne deposition of contaminated dusts from fugitive dust sources in and adjacent to the mining/smelting complex. Air monitoring information collected during RI/FS activities and summarized in the RADER indicates that airborne dusts transported into the Populated Areas have concentrations ranging from 1,000 to 20,000 ppm lead.

Total dry airborne particulate deposition rates average 2,532 ug/m2/hr and 1,768 ug/m2/hr at the Smelterville Mine Timber and Kellogg Middle School monitoring sites, respectively (Figure 52). Wet deposition rates averaged 484 and 487 ug/m2/hr at the Smelterville and Kellogg sites, respectively. More than 80 percent of the total particulate and more than 90 percent of most metals deposition occurs as dry deposition. The maximum dry deposition rate observed was 12,595 ug/m2/hr at the Mine Timber site during the second week of September 1988. Only four metals were observed to have dry deposition rates consistently exceeding 1.0 ug/m2/hr. Those were iron, lead, manganese, and zinc with annual average deposition rates at the Mine Timber site of 132, 12.7, 8.6, and 11.3 ug/m2/hr, respectively. The maximum weekly lead deposition rate observed was 83.8 ug/m2/hr at the Mine Timber site, also occurring during the second week of September.

The highest deposition rates were observed during the weeks that also included the servere dust event days with Total Suspended Particulates (TSP) >150 ug/m3 shown in Table 5-9. The 1988 data confirm that both total solids and contaminant particulate deposition seem to be event-related in a manner similar to the TSP and ambient air metals concentration discussed in the last section. At both sites, more than 25 percent of the total annual solids deposition occurred in four individual weeks in 1988. Those included 1 week in each of May, August, September, and October. The same weeks accounted for 31 percent of total lead, 18 percent of total cadmium, and 29 percent of total arsenic deposition. The 1988 seasonal data also showed a frequency and magnitude of severe dust events (TSP >300 ug/m3) similar to that observed in 1987, but absent in 1989.

Table 5-9 Individual Filters with TSP > 150 ug/m-3 November 1987 to November 1988
(See Data in Original Document)

These results suggest that deposition, similar to TSP, is eventrelated with the bulk of deposited solids and meals coming as a result of high wind speeds impacting barren dust sources in the vicinity of the monitors.

Water erosion of hillsides near the smelter complex is a migration pathway to

residential soil, particularly in yards abutting hill slopes. Mass loading rates are high along these steep barren locations where sheet and rill erosion with gullying are significant. Metals contents on the hillsides average 5,000 ppm lead.

Lead leachability from residential soils was determined by Extraction Procedure (EP) Toxicity, and Toxicity Characteristic Leaching Procedure (TCLP) analyses. These tests are used to determine if a material should be considered a hazardous waste pursuant to the Resource Conservation and Recovery Act (RCRA) and, consequently, subject to RCRA storage and disposal requirements. Results showed 3 out of 23 EP Toxicity samples exceeded the RCRA lead threshold level of 5 ppm. Two of the six TCLP samples exceeded the threshold levels for lead.

6 SUMMARY OF SITE RISKS

6.1 HUMAN HEALTH RISKS

The RADER presents a detailed discussion of the risk assessment for the Populated Areas. In the RADER, both carcinogenic and noncarcinogenic effects of contaminant exposures are evaluated. A Non-populated Areas risk assessment is being conducted in concert with the Non-populated Areas RI/FS.

6.1.1 EXPOSURE ASSESSMENT

The contaminants used in the exposure evaluation and risk assessment are all metals that exhibit: 1) elevated concentrations in residential soils and dusts relative to background concentrations; 2) decreasing concentrations in environmental media with increasing distance from the industrial complex; and 3) potential for human toxicity following incidental and chronic exposures. Contaminants of concern include antimony, arsenic, cadmium, copper, lead, mercury, and zinc.

Receptor populations at risk are identified as the current and past residents of the Populated Areas of the site. Three groups have been evaluated in terms of contaminant exposures and consequent risks. These are:

1) A general population of residents that are assumed to live, since birth, under the conditions represented by the contamination levels found since 1983 for a 70-year lifetime (referred to as the current scenario which would also be a future scenario under the No Action Alternative)

2) A general population of residents who were born in 1971 and were 2 years old during the period of maximum exposure onsite and who remain onsite under current conditions for a 70-year lifetime (referred to as the historical scenario)

3) A sensitive subpopulation of children exposed to lead

Historical exposures, since 1971, were evaluated because of documented high

contaminant concentrations during 1973-1975. Airborne lead concentrations were approximately 100 times greater during this period than current levels. Consideration of these exposures is critical for evaluating the potential chronic risks of metal contaminants on the population.

Both the current and historical populations (numbers 1 and 2 above) are representative of baseline conditions--those conditions under which no remedial action has been undertaken (the No Action Alternative).

The principal exposure media and associated receptor pathways characterized for the evaluation of base-line human health risk for the typical resident in the Populated Areas of the Bunker Hill site are:

- o Ingestion of residential surficial yard soils
- o Ingestion of house dusts
- o Inhalation of air particulate matter
- o Consumption of national market basket variety produce (foodstuffs available on supermarket shelves representing food of average consumers) and water ingestion from public water supplies (public water is supplied from a surface water source outside site boundaries)

Additional exposures that could be experienced by members of the population who engage in potentially high-risk activities are evaluated as incremental exposures. The following incremental exposures were evaluated:

- o Consumption of contaminated local groundwater
- o Ingestion of other soil/dust at extreme (95th percentile concentration) residential soil and house dust concentrations
- o Ingestion of extreme amounts (1 gm/day) of soil and dust during childhood (typical of "pica-type" behavior)
- o Consumption of local fish from the Coeur d'Alene area
- o Consumption of local vegetable garden produce
- o Inhalation of out door air particulate matter during episodic, high wind events

To determine an individual's level of risk resulting from participation in potentially high-risk activities, the appropriate incremental risk(s) were added to the baseline estimate. If an individual does not engage in any of the incremental activities evaluated, then the risk to that individual would be the baseline estimate. The incremental exposure analysis can be used to determine the Reasonable Maximum Exposure scenario for the Populated Areas.

Exposure and consequent risks were evaluated for each of the two baseline periods (current and historical) in three separate areas (Smelterville, Kellogg/Wardner/Page, and Pinehurst) for the average or typical population. The risk assessment was completed assuming current land uses would continue to be residential.

Lifetime or chronic exposures were evaluated for the typical resident by estimating contaminant intakes using avaerage media concentrations (see Table 6-1). For this evaluation, arithmetic mean concentrations for exposure media were used to represent average or typical long-term exposure levels. For residential soil and house dust exposures, geometric mean concentrations were calculated and used for evaluating typical long-term exposures. Geometric mean values for these media are expected to be more representative of average expsoures because of the statistical distributions exhibited by soil and house dust metal concentrations.

Table 6-1 Contaminants Evaluated, Exposure Routes and Sources, and Exposure Scenarios Addressed in the Risk Assessment

(See Data in Original Document)

Chronic exposures at extreme levels are not expected for the typical resident. Therefore, chronic exposures to extreme concentrations of site contaminants are not evaluated in the baseline chronic assessment. Extreme media concentrations represented at 95th percentile levels were evaluated as incremental and subschronic exposures.

The traditional approach for risk characterization associated with lead exposure is currently inappropriate because an acceptable Reference Dose (RfD) for lead is not available. Therefore, risk characterization for subchronic lead exposure was accomplished by using observed childhood population blood lead levels and environmental media lead concentrations collected over the last 17 years in an integrated uptake/biokinetic doseresponse model. The model was used to relate childhood blood lead levels to contaminated media exposures. Model inputs and criteria were selected and validated using site-specific data as described in the RADER.

Table 6-1 presents a summary of contaminants of concern, exposure routes and sources, and scenarios addressed in the exposure evaluation and risk assessment.

6.1.2 TOXICITY ASSESSMENT

A detailed discussion of the toxicity of site contaminants is presented in Section 3.5 of the Protocol Document. Table 6-2 provides a summary of the most sensitive effects for each of the seven site contaminants of concern.

Table 6-2 Summary of Most Sensitive Adverse Health Effects of Site Contaminants of Concern

(See Data in Original Document)

Tables 6-3 and 6-4 summarize the available Cancer Potency Factors (CPFs) and Reference Doses (RfDs) for the site contaminants of concern. These values were obtained from the Health Effects Summary Tables and Integrated Risk Information System.

Table 6-3 Available CPFs for Site Contaminants of Concern
(See Data in Original Document)

Table 6-4 Noncarcinogenic Effects and Associated RfDs for Site Contaminants of Concern

(See Data in Original Document)

6.1.3 RISK CHARACTERIZATION

6.1.3.1 Carcinogenic Risk

Excess lifetime cancer risks are determined by multiplying the intake level with the cancer potency factor. These risks are probabilities that are generally expressed in scientific notation (e.g., 1x10E-6). An excess lifetime cancer risk of 1 x 10E-6 means that if a population of 1 million people were exposed to the baseline condition over a 70-year lifetime, it is expected that there would be one additional cancer above the cancer events due to others causes. The current U.S. cancer rate is one in four. Therefore, in a population of 1 million people, 250,000 cancer events are predicted. Under a 10-6 risk scenario, 250,001 cancer events would be predicted.

Results of the chronic exposure and risk characterization indicate that excess (above background) carcinogenic risk is associated with baseline exposures and consequent intakes for arsenic and cadmium in air. Total baseline (70-year lifetime) risk to lung cancer, due to inhalation of arsenic and cadmium under current site conditions, is from 2 to 32 times greater than for offsite background. Under the historical scenario, risk to lung cancer was two to six times greater than the current scenario for the same communities. Baseline cancer risk estimates indicate that the typical population exceeds U.S. EPA's acceptable range for cancer risk (10E-4 to 10E-6).

Acceptable levels of risk to lung cancer may never be attained at any future arsenic and cadmium air levels for those individuals who have had considerable historical and cumulative exposures. Tumor registry data support the presence of a disease-causing agent for the increased occurrence of respiratory cancers in the area.

Baseline carcinogenic risk due to site exposures is approximately 30 percent greater than background carcinogenic risk (9.8 x 10E-4). Baseline carcinogenic risk in conjunction with the consumption of site groundwater in Smelterville and Kellogg due to arsenic intakes could result in a doubling of the risk associated with background exposures. Excess health risk due to arsenic in groundwater makes this source unsuitable for drinking in many

areas of the site. Groundwater is not currently used as a municipal drinking water source.

Table 6-5 presents a summary of the baseline and incremental carcinogenic risk estimates.

Table 6-5 Summary of Baseline and Incremental Carcinogenic Risk Estimates
(See Data in Original Document)

6.1.3.2 Noncarcinogenic Risk

Potential concern for noncarcinogenic effects of a single contaminant in a single medium is expressed as the hazardous quotient (HQ). By adding the HQs for all contaminants within a medium or across all media to which a given population may reasonably be exposed, the Hazard Index (HI) can be generated. The HI provides a useful reference point for gauging the potential significance of multiple contaminants exposures within a single medium or across media. Excess risk is determined to be where the HI is greater than or equal to 1.0.

All estimated baseline noncarcinogenic risks for specific toxic endpoints and target organs resulting from oral intakes of site contaminants of concern have been determined to be acceptable (HI < 1).

Potential activities that could result in unacceptable risk to noncarcinogenic disease are associated with metal intakes resulting from consumption of site groundwater, excessive soil and dust ingestion by children, and consumption of local garden produce.

Table 6-6 presents the summary of excess risks evaluated in the noncarcinogenic risk assessment.

Table 6-6 Summary of Exposure Routes, Scenarios, and Potentially High-Risk Activities That Could Result in Unacceptable Chronic Risk to Noncarcinogenic Disease
(See Data in Original Document)

6.1.3.3 Subchronic Exposure

The most recent lead health survey of area children indicates that current blood lead levels for many children exceed levels at which adverse health effects are associated. In 1990, 2 of 362 children had blood lead levels exceeding 25 ug/dl. Fifty percent (50%) of the children within an approximate 2-mile radius of the industrial complex had blood lead levels exceeding 10 ug/dl. Thirty percent (30%) of the children within the 2- to 3-mile radius of the industrial complex had blood lead levels exceeding 10 ug/dl.

CDC's 1985 Health Advisory for Blood Lead Levels states that "a blood lead level in children of 25 ug/dl or above indicates excessive lead absorption and constitutes grounds for medical intervention." Recent information indicates that adverse health effects are associated

with blood lead levels at 10 to 15 ug/dl, or possibly lower. CDC is expected to establish 10 ug/dl as the level above which action should be taken. In addition, ATSDR is supportive of the goal of reducing childhood blood lead levels to below 10 ug/dl.

A review of past exposures and health survey data at the Bunker Hill site indicates that during extreme exposures in the early to mid-1970s, up to 80 percent of the children exhibited blood lead levels that are associated with adverse neurobehavioral development that persists into young adulthood. Additional concern for past lead exposures (prior to smelter closure in 1981) is due to the potential release of lead from normal bone resorption during pregnancy and lactation and the resultant pre- and post-natalexposures to children who are born today of mothers who were exposed as children in the 1970s.

Subchronic exposures and consequent intakes could increase health risks in the short term to levels well above those estimated for baseline chronic risks. Ingestion of extreme amounts of soil and dust during childhood (ages 2 to 6 years), characterized as "pica-type" behavior, could yield up to 10 times greater metal intakes than for the typical child. These extreme intakes due to soil/dust ingestion could amount to approximately 2 mg Pb/day, resulting in dangerous blood lead increases in young children. "Pica-type" behavior could present extreme risk to this highly susceptible sub-group of the population, and requires control if observed.

Consumption of local garden produce can yield extreme intakes of cadmium, lead and zinc. Up to 220 times as much lead can be ingested from the consumption of local garden vegetables grown in Smelterville and Kellogg versus that associated with the consumption of national market basket variety produce. Children and pregnant women (as surrogates to the fetus) are most susceptible to the adverse effects associated with consequent lead intakes. Up to 62 times as much cadmium can be consumed in local garden produce versus market basket variety produce, thus presenting unacceptable chronic and subchronic risk to renal disease.

6.1.4 HUMAN HEALTH RISK SUMMARY

In summary, the conclusions of the RADER state that current site conditions present an environment where there are excessive risks associated with several different exposure pathways. These are:

- Carcinogenic risk associated with exposure to:
  - Arsenic via potential groundwater consumption
  - Arsenic and cadmium via inhalation
- Chronic noncarcinogenic risk associated with exposure to:
  - Arsenic, cadmium, and zinc via potential groundwater consumption
  - Antimony, cadmium, mercury, and lead via excessive soil and dust

ingestion (characterized by "pica-typed" behavior)

- Cadmium and lead via local garden produce consumption

- Subchronic noncarcinogenic risk associated with exposure to:
  - Lead via ingestion of soil and dust
  - Cadmium, lead, and zinc via local garden produce consumption

Subchronic lead absorption among young children is the most significant health risk posed by this site.

The major routes for lead absorption are:

- Ingestion of contaminated soils in residential yards and other residential environs
- Ingestion of contaminated house dusts that are resultant from tracking of residential soils and deposition of airborne particulate
- Inhalation and ingestion of airborne particulate matter derived from fugitive dust sources throughout the site

6.1.5 THE 1,000 PPM THRESHOLD CLEANUP LEVEL

A remedial action objective for this operable unit is to decrease the exposure to lead-contaminated residential soils such that 95 percent or more of the children in the area have blood lead levels below 10 ug/dl and that less than 1 percent have blood leads greater than 15 ug/dl. The 1,000 ppm lead cleanup threshold level selected for yard soil remediation at Bunker Hill is a site-specific and media-specific value chosen to meet these objectives. This level is not a target exposure concentration. Rather, it is the maximum soil lead level that any child may be exposed to in his or her home yard. This should not be construed to suggest that this level is health protective for soils at other sites, or other soil and dust media at the Bunker Hill site. A child living on an unremediated yard of 1,000 ppm is estimated to have a 0.1 to 2.5 percent (depending on various assumptions) chance of exceeding 15 ug/dl blood lead in the Bunker Hill post-remediation environment. The following are several reasons why this solution applies only for residential yard soils and only at this particular site:

Response Rate: The response rate value for this site was arrived at after extensive review of epidemiologic and environmental data collected at the site for more than 15 years. Analyses of those data suggest that the dose-response relations between contaminated soils and dusts and resultant blood lead levels in children is about half that observed at other lead-contaminated sites. Whether the lesser response rate is due to reduced intake (lower soils and dust ingestion rates) or reduced uptakes (lesser absorption of ingested lead in soils) cannot be discerned from the data. The selection of the 1,000 ppm threshold level assumes the latter

(i.e., reduced absorption rates at this site).

Total Lead Intake: Predicted blood lead level resultant from remedial activities are based on total lead intake from all media. The four principal pathways are lead in diet, drinking water, air, and soils and dusts. The effectiveness of the 1,000 ppm threshold level for yard soils is dependent on several assumptions regarding reduced intakes along other pathways. Some of those assumptions are based on assessments of other remedial activities on the site and substantial reductions in dietary intake achieved from nationwide lead reduction initiatives. Those assumptions may not apply to other sites.

Composite Soil/Dust Lead Concentrations: Analyses presented in the RADER suggest that the composite concentrations of lead in all the soils and dusts ingested by children must be reduced to 700 to 1,200 ppm at this site to meet the remedial action objective of less than 5 percent of children having a blood lead of greater than 10 ug/dl. There are several contributing sources to this overall soil and dust loading. Those include yard soils, house dusts, road dusts, play area soils, fugitive dust sources, and other soils in the community where children may congregate. Residential yard soils are an important component of the overall soil and dust loading. A substantial portion of children's exposure results from direct contact in the yard. A substantial portion of house dust loading results from yard soils transported into the home and additional children's exposure results from visits to yards other than their own home. Yard soils may also be a source of contaminated dusts circulating through the community via air, water, and mechanical pathways. Removing all yard soils greater than 1,000 ppm will have positive effects along all these pathways and routes of exposure. However, achieving the remedial action objectives will require additional activities among the soil and dust sources other than yard soils. Those actions are specific to this site and may not be applicable to other locales.

Distribution of Yard Soil Lead Concentration: The effectiveness of the cleanup strategy in meeting remedial action objectives depends on the post-remediation distribution of contaminant levels. That distribution will be site-specific and, likely, inapplicable to other locations. The imposition of the 1,000 ppm cleanup threshold at the Bunker Hill site will result in remediation of more than 75 percent of the yards in most residential areas. The mean yard soil lead concentrations in area communities will be reduced from nearly 3,000 ppm to less than 200 to 300 ppm. This represents a tremendous reduction in total environmental lead loading in the community and should have positive effects in other media as well. Substantial benefit will result in the form of reduced exposure from several sources.

Actual or threatened releases of hazardous substances from this site, if not addressed by implementing the response action selected in this ROD, may present an imminent and substantial endangerment to public health, welfare, or the environment.

6.2 ENVIRONMENTAL RISKS

This Record of Decision addresses the remediation of residential soils within the Populated Areas of the Bunker Hill Superfund Site. There are no critical habitats or endangered species or habitats affected by residential soils contamination or anticipated effects caused by

future remediation. An ecological risk assessment is being conducted as part of the Non-populated Areas RI/FS.

The urban component of the ecosystem at Bunker Hill has been impacted by historical mining and smelting activities. The average heavy metal concentrations in residential soils and community road shoulders are higher than on the hillsides portion of the site. Many of the residential soils have metal concentrations capable of inducing toxicological effects on soil micro-organisms, invertebrates, and plants. Comparative concentrations in various other soil types have resulted in reduced productivity, yields, decomposition, and nutrient cycling rates. Other animals that inhabit the urban areas such as field mich and squirrels, as well as cats and dogs, are susceptible to ingestion of residential soils with an increased risk of chemical stress.

Management of soil and vegetation at Bunker Hill can facilitate natural and favorable conditions within the urban ecosystem by reducing the mobility of contaminants and their potential for inducing chemical stress. The replacement of residential soils and vegetation is expected to enhance the micro-habitat niches for the flora and fauna that use them.

7 DETAILED DESCRIPTION OF ALTERNATIVES

This proposed cleanup action involves residential yards, an area that is typically used for many different activities and purposes. While it is important that the cleanup action block the routes by which people come in contact with contaminants in the soil, it is also important that the cleanup action allow residents to use their yards for their many purposes. For example, while a concrete or asphalt layer would block the pathway between the contamination and residents, it would make it impossible for residents to use their yards for typical activities, such as planting and gardening. Therefore, except for the No Action Alternative, all of the alternatives are designed to reduce human exposure to contamination, while maintaining the integrity of the individual yards.

7.1 ALTERNATIVE 1--NO ACTION

The No Action Alternative provides a baseline for comparing against other alternatives. The site would be left in its current condition. Existing institutional controls, such as the Health Intervention Program, would be discontinued. Because no remedial activities would be implemented with the No Action Alternative, long-term human health and environmental risks from residential soils at the site would be essentially the same as those identified in the RADER:

- Significant health risks to young children associated with exposure to ingestion of contaminated soil, ingestion of contaminated house dusts, and inhalation and ingestion of airborne particulate matter would maintain currently unacceptable health conditions and could result in dangerous blood lead increases in young children.

- Excessive soil and dust ingestion by "pica-type" children could result in toxic effects due to antimony, cadmium, and lead.

o Consumption of local produce can increase intakes of cadmium, lead, and zinc, resulting in neurological and renal disease.

Unacceptable high blood lead concentrations in some children would probably continue and the potential of increases in blood lead concentrations could increase because of the termination of the health intervention program.

Environmental monitoring would be conducted under the No Action Alternative. Environmental monitoring would occur for the following media:
If the columns or type in the table(s) below appear misaligned, please press Ctrl+V and scroll right to view. When done, press ESC to restore your original view.

Media	Parameters
Air	Suspended particulates, Pb and As concentrations
Residential Soils	Contaminant metals concentrations

Sampling locations would be consistent with previous sample collection sites to provide a basis for historic comparison. In addition to monitoring environmental media it is expected that childrens' blood would continue to be screened for lead.

7.2 COMMON COMPONENTS OF ALTERNATIVES 3--VARIABLE CUT/REMOVE/FILL/DISPOSAL; 5--SOD REMOVAL/SOD REPLACEMENT/DISPOSAL; 6--DEEP REMOVAL/FILL/DISPOSAL; AND 8--VARIABLE CUT/REMOVE/FILL/TREAT/DISPOSAL

All of the remaining alternatives have components in common (use of institutional controls, revegetation, dust suppression, excavation/backfill, extent of remediation, disposal, and monitoring). Although the description of these components is not repeated in the discussions for each alternative, differences in their planned implementation are identified where appropriate. ARARs for all alternatives are similar and are discussed in Section 10. Each of these common components is discussed below.

7.2.1 INSTITUTIONAL CONTROLS

Institutional controls would be implemented to a certain degree with each alternative. The reliance on institutional controls is dependent on the remedial action technologies employed and their long-term effectiveness in protecting human health and the environment. The detailed evaluation of the proposed institutional controls are included in the document entitled An Evaluation of Institutional Controls for the Populated Areas of Bunker Hill Superfund Site, which is part of the Residential Soils Administrative Record.

The range of institutional controls consists of the following components:

- o Deed notices
- o Public education
- o Excavation regulations and permits
- o Health intervention program
- o Contaminated soil collection system
- o Clean soil supply system
- o Post-cleanup administration and evaluation
- o Sod maintenance ordinances
- o Lawn maintenance contracting

7.2.2 REVEGETATION

Revegetation of residential yards is a component of each alternative. The lawn areas of remediated yards would generally be revegetated with sod. Steep hillsides and other remediated areas not currently planted with lawns (such as vacant lots) would be stabilized and hydroseeded with native grasses. Native grasses require less maintenance and are more tolerant of the local climatic conditions. If preferred by a property owner, hydroseeding with native grasses could be substituted for the sod. To the extent practicable, all yard landscaping would be returned to its original condition.

7.2.3 DUST SUPPRESSION DURING REMEDIATION

Dust suppression measures would be implemented throughout the remediation process to reduce exposure of workers and residents to airborne contaminants. Dust suppression would include:

- o Watering of residential yard areas prior to excavation activities
- o Continued watering during excavation, as necessary
- o Placement of tarps or covers over excavated materials
- o Use of tarps or covers over truck beds to reduce blowing dust and spillage during transportation to the waste repository
- o Daily cleanup of all spilled or tracked soils from sidewalks, roadways, etc.

Appropriate air monitoring would be conducted to identify the occurrence of contaminant migration during remedial activities. Any exceedances of the standards would result in immediate implementation of additional dust suppression measures or a shutdown of construction activities.

7.2.4 EXCAVATION/BACKFILL/COVER

For all alternatives, remediation of residential yards would be completed by either covering with a layer of uncontaminated soil or by removing and replacing contaminated soil or sod with uncontaminated materials.

A range of alternatives was developed to provide decisionmakers with several options. Alternative 5 is an option with minimal soil removal and replacement. A 12-inch removal and replacement is presented in Alternative 3. A 6-inch soil barrier was considered during the development of Alternative 3. However, it was concluded that a 6-inch depth is insufficient to provide a viable option as a barrier technology in a residential area, if the underlying material is contaminated. This is because a 6inch barrier could be penetrated by such common occurrences as a digging dog, a homeowner planting bulbs, or children's play activities. To complete the range of alternatives, Alternative 6 was developed to evaluate deep removal of contaminated materials.

7.2.5 EXTENT OF REMEDIATION

For all of the alternatives, the areal extent of remediation would be consistent. For each residential yard, the exact nature of the remediation (e.g., how much sod to replace, which bushes to remove, etc.) would have to be considered on a case-by-case basis. However, for consistency, the following areas would generally be remediated within each yard:

- Sod areas
- Roadway shoulder (if curb and gutter is not present) to the extension of the lot lines
- Alleys (if unpaved) to the extension of the lot lines
- Planters and other landscaped areas
- Garden areas
- Unpaved driveways
- Garages with dirt floors
- Storage areas

In short, remediation would occur in any area within and adjacent to the residential yard where children could play and could potentially come in contact with contaminated soils. Areas that currently provide a barrier from the underlying soils (such as paved sidewalks and driveways) would not require remediation.

7.2.6 DISPOSAL

The proposed site for disposal of contaminated residential soils for all alternatives is the Page Ponds tailings impoundment. Page Ponds is an old tailing impoundment that is currently the site of the South Fork Coeur d'Alene Sewer District treatment facility. On either side of the sewage lagoons are "benches" that are primarily tailings, denuded of vegetation, and consequently are a source of windblown dust to the valley. The benches (east and west dikes) is the area recommended for the residential soils repository. Consolidation of residential soils and sod onto the Page benches will contribute to reducing fugitive windblown dust throughout the valley.

Since the volume of material requiring disposal will vary with the selected alternative, the volume of soil wastes may exceed the capacity of the Page benches. In that case, an additional disposal site will need to be used to supplement the disposal capacity of Page Ponds since the approximate capacity of Page Ponds is 860,000 cubic yards.

The disposal site will have an impermeable cap or cover (i.e., one that is designed to minimize migration of contaminants) placed during closure. The long-term management of the area will include maintenance of the cover and groundwater monitoring. In addition, access restrictions and land use restrictions and/or notices will be used to ensure that future use of the property is not incompatible with residential soils repository.

7.2.7 ENVIRONMENTAL MONITORING

Regardless of the alternative selected, contaminated materials will remain within the residential areas of the site. Alternative 6, which requires deep excavation to remove materials, will most likely not remove all contaminated material. Therefore, environmental monitoring will be continued at the site for an indefinite period. It is estimated that environmental monitoring of fugitive dust and residential soil and litter would continue. Monitoring will occur at previous sampling locations to provide a basis or historical comparisons. It is expected that blood lead levels would also be monitored. For cost estimating purposes, it is assumed that a greater extent and frequency of sampling will be required in Alternative 5 than the other alternatives, since it would place only a sod layer barrier between the contaminants and the residents.

7.3 ALTERNATIVE 3--VARIABLE CUT/REMOVE/FILL/DISPOSAL

Alternative 3 consists of the following options:

- o A 2-inch gravel barrier and 10-inch cover without soil excavation

- o A 2-inch gravel barrier installation, and a 10-inch soil replacement after excavation and removal of up to 12 inches of soil (yards would be above grade for excavations less than 12 inches)

Both options are similar in that each incorporates a combination of a visual barrier and a separate soil cover. They differ in where they can be applied to a residential yard because of drainage and homeowner considerations. Whatever the excavation depth, this alternative will result in the placement of a minimum of 12 inches of clean material.

The option of a gravel/soil cover barrier without additional soil excavation is preferred because it minimizes the volume of contaminated soil requiring disposal. A 2-inch clean gravel layer with a 10-inch soil cover would be selected for implementation at residences in which the foundation is high enough in relation to existing grade to allow its use, where permission is granted by the respective property owner, and at residences where drainage is not a problem.

The cover would consist of 2 inches of clean gravel overlain by 10 inches of clean topsoil from an offsite borrow source. The gravel layer would provide a visual and physical barrier indicating to the landowner that the bottom of the remediated soils had been reached, isolating the underlying contaminants from inadvertent exposure. Also, the gravel layer would act to some degree as a capillary barrier to the subsurface migration of metals. Clean fill would be revegetated by sodding. To the extent practicable, the yard landscaping would be returned to its original condition.

A 24-inch layer of topsoil would be placed in established garden areas since some plant roots and tubers extend below 12 inches, but generally less than 24 inches. Future activities that penetrate the 12-inch cover, such as utility line installation, planting of larger trees and shrubs, and basement or foundation excavation, would be controlled through ordinances regulating excavation, as detailed under Section 7.2.1, Institutional Controls.

For those residences in which a simple gravel barrier/soil covering cannot be implemented, contaminated soils would be excavated and replaced with a clean gravel/topsoil barrier. Various depths of excavation and fill would be necessary based on site conditions:

- o Excavate 12 inches; replace with 2 inches of gravel and 10 inches of soil.

- o Excavate less than 12 inches; replace with 2 inches of gravel and 10 inches of soil (finished grade would be above existing grades).

- o Excavate 24 inches, replace with 2 inches of gravel and 22 inches of soils (for established garden areas).

The choice of excavating to less than 12 inches is dependent upon the yard grade in relation to the house floor grade and depth of contamination. Under most circumstances,

building codes do not allow yard grades to be higher than house floor grades. The next step to implementing this alternative would be to excavate soils to the selected depth below the ground surface. All sod or other surface coverings, except for pavements, would be removed and disposed of along with the soil. Large trees (4-inch diameter and larger) and shrubs (taller than 3 feet) would be saved, if possible. Trees and shrubs left in place would be trimmed back and contaminated soil would be removed by hand from around the roots. The "clean" soil used to replace the excavated soil would meet borrow source and landscaping specifications. Backfilled areas that were previously lawn areas would generally be revegetated with sod. In some backfilled areas it may be more appropriate to revegetated using hydroseeding with native grasses (steep hillsides, vacant lots, etc.) To the extent practicable, however, the yard landscaping would be returned to its original condition.

The volume of material to be disposed is estimated to be 640,000 cubic yards.

Regardless of the option employed under Alternative 3, environmental monitoring of fugitive dust, residential soils, house dusts, and periodic blood lead analyses of residents would be continued. Monitoring would occur at previous sampling locations to provides a basis for historical comparison.

7.4 ALTERNATIVE 5--SOD REMOVAL/SOD REPLACEMENT/DISPOSAL

Alternative 5 consists of contaminated sod removal and replacement.

Residential yards would be cleared and grubbed, which includes removal of sod, brush, and stumps. Alternative 5 would not include any removal of contaminated soils or replacement with clean soils in grassed areas. The clean sod would be placed over the top of contaminated soils. To the extent practicable, the yard landscaping would be returned to its original condition.

All areas not to be covered with new sod would be remediated using excavated/replace/dispose techniques. Areas such as planters and graveled area would be excavated to 6 inches. Garden areas would be excavated to 24 inches and backfilled with clean soil, similar to Alternative 3. Contaminated materials would be disposed of in the Page Ponds Repository. The estimated volume for disposal would be 203,500 cubic yards. Clean fill from an offsite borrow source would be used to replace the excavated materials.

Future activities that penetrate the clean sod layer, such as utility line installation, planting of trees and shrubs, and basement or foundation excavation, would be controlled through ordinances regulating excavation, as detailed under Section 7.2.1, Institutional Controls. Additional institutional control would have to be implemented with Alternative 5 to maintain the long-term viability of the sod layer. These controls would include ordinances requiring homeowners to water and maintain the replacement sod to an acceptable level. Additional inspection would be required by the various government entities to ensure that the sod maintenance ordinances were effectively enforced. A professional lawn maintenance company would be retained to advise and assist the homeowners with proper sod maintenance. The lawn maintenance company would also provide and apply the necessary fertilizers and chemicals to

ensure the health and vigor of the sod barrier. Environmental monitoring after remediation would be continued.

7.5 ALTERNATIVE 6--DEEP REMOVAL/FILL/DISPOSAL

Alternative 6 includes removal of contaminated soil to a depth of 7 feet and replacement with clean material. Although this is a deep removal, there may be contaminants left inplace in some areas.

The institutional controls requirement with this alternative would be considerably reduced. Since contaminated residential soils would be removed to a depth of 7 feet, future institutional controls for residential yards would be minimized. The public information and health intervention programs would be required, but at a reduced level. Environmental monitoring would be continued.

For residential yards, all contaminated soils would be excavated and replaced with clean soil. The depth of excavation would be determined on a site-by-site basis. The excavation would extend to a depth at which the threshold level was reached or to approximately 7 feet.

Prior to excavation activities, the depth and concentration of lead contamination would be determined in areas to be remediated. Selection of sampling strategy and depth of soil removal would be a function of the remedial design/remedial action process.

Once excavation and fill depths are selected, the next step to implement this alternative would be to excavate soils to the selected depth below the ground surface. All sod or other surface coverings would be removed and disposed of along with the soil. The need to remove and replace pavements and sidewalks would be determined on a case-by-case basis. All trees and shrubs would be removed. The soil used to replace the excavated soil would consist of clean soil from an offsite borrow source. Backfilled areas would be revegetated. To the extent practicable, the yard landscaping would be returned to its original condition.

Soil, sod, and other materials that are removed would be disposed at an appropriate disposal site. It is estimated that Alternative 6 would generate 4.45 million cubic yards of wastes. Preliminary estimates indicate that approximately 860,000 cubic yards of wastes could be disposed of at the Page Ponds Repository. This means that approximately 3.6 million cubic yards of wastes would have to de disposed of at another site, if Alternative 6 is implemented.

Special care would have to be taken when excavating near foundations, basements, and utilities to avoid damage to existing structures and facilities. Temporary shoring and supports may be required. It may be advantageous to remove and replace utility lines, rather than shore and support them during construction.

Because of the inconvenience to the residents and potential liabilities associated with this alternative, the residents would be temporarily relocated during construction. The relocation would be to local motels or hotels and would be expected to last 2 to 3 weeks for an

average residential yard remediation.

7.6 ALTERNATIVE 8--VARIABLE CUT/REMOVE/FILL/TREAT/DISPOSAL

Alternative 8 identical to Alternative 3 except that the excavated soils would be treated with pozzolanic agents prior to disposal.

In Alternative 8, excavated soils would be mixed with pozzolanic agents in a pug mill prior to disposal. The addition of pozzolanic agents will tend to solidify contaminated soils and may reduce contaminant mobility. If this alternative is chosen, treatability studies would be conducted to determine if these soils are amenable to pozzolanic fixation, and if pozzolanic fixation will adequately reduce contaminant mobility. Environmental monitoring would be continued at predetermined intervals. The volume of material to be disposed would increase approximately 50 percent from 640,000 cubic yards to 960,000 cubic yards as a result of pozzolanic treatment.

8 COMPARATIVE ANALYSIS OF ALTERNATIVES

A comparative analysis of alternatives using each of the nine evaluation criteria, as required by federal regulation, is presented in this section. The purpose of this analysis is to identify the advantages and disadvantages of each alternative relative to the other alternatives. A separate evaluation of the alternatives is presented under the heading of each criterion.

8.1 PROTECTION OF HUMAN HEALTH AND ENVIRONMENT

Protection of human health and the environment is addressed to varying degrees by the five proposed alternatives. Alternative 1 is the No Action Alternative. As proposed, it would have no effect on the site; therefore, it does not address any of the identified concerns. Indeed, an increase in blood lead concentrations over time could occur.

Alternative 3, 6, and 8 provide protection of human health through installation of a soil and sod barrier between residents and underlying contaminated materials. All three address the concerns of exposure through direct contact with soil contaminants or tracking contaminated residential soil into homes as a source of house dust. Alternative 5 addresses these concerns, but to a lesser extent than the others because of the requirement for rigorous maintenance. All alternatives address the exposure pathway of local garden produce.

None of the alternatives would alter the toxicity or persistence of the soil contaminants. Alternative 8 does include a treatment plan for excavated soils that would solidify the soils once they are removed from the site and may reduce mobility.

In general, permanence of remedial actions is greatest for Alternative 6 with its essentially complete removal of contaminated soils. Alternatives 3 and 8 provide a degree of permanence through removal of surficial layers of contaminants, requiring less implementation time and effort, but they rely on a greater need for institutional controls. Alternative 5 provides

the least amount of protection on a permanent level because of its reliance on institutional controls and the susceptibility of the sod layer to withstand normal human activities and inconsistencies in maintenance.

8.2 COMPLIANCE WITH APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARs)

With the exception of Alternative 1, the No Action Alternative, all alternatives meet federal and State of Idaho ARARs. A further discussion of compliance with federal and state ARARs is included in Chapter 10.

8.3 LONG-TERM EFFECTIVENESS

The residual risk (the risk remaining after implementation) increases from lowest to highest in the following order of alternatives: 6, 3 and 8, 5, and 1 (No Action Alternative). Alternative 6 would result in the least amount of residual risk because of the volume of contaminated soils that would be removed to ensure that future exposure to onsite residential soil sources does not occur. Although Alternatives 3 and 8 do not reduce residual risk to the same level as Alternative 6, they would protect the communities in the long term if institutional control measures were implemented and followed. Alternative 5 provides the least long-term protection since the sod barrier may be easily breached.

Maintenance requirements for all alternatives would be fairly similar. Each alternative incorporates a sod or grass cover and similar institutional controls. However, the level of the requirement varies with the alternative. Alternative 5 is more sensitive to maintenance requirements because a layer of sod is the only barrier between residents and the underlying contaminated soils. Alternatives 3 and 8 follow with a layer of clean fill of at least 12 inches under the sod layer. Alternative 7 requires the least amount of maintenance as a result of the extensive layer of fill (up to 7 feet) needed to return residential yards to their original grade.

Environmental monitoring would vary according to the degree of protectiveness incorporated within the remedial alternatives. Alternative 5 would require the greatest amount of monitoring to ensure that the sod barrier remains effective. This would entail frequent soil and litter metals analyses and blood lead analyses. Alternatives 3 and 8 would require periodic monitoring of the surficial soil layer to check for airborne recontamination and periodic monitoring of the remediated soil profile to check for disruption and recontamination of the soil barrier. Alternatives 3 and 8 would also require periodic blood lead analyses. Alternative 6 would require periodic monitoring of the surficial soil layer and periodic blood lead analyses. Alternative 1 would include environmental monitoring to check for changes in contaminant levels with time. Blood lead screening would be discontinued when warranted.

The disposal recommendation for residential soil is consistent for all alternatives except for Alternative 8, which includes the addition of pozzolanic agents prior to disposal. The long-term effectiveness of the disposal recommendation is ensured through appropriate closure requirements and management by institutional controls.

8.4 REDUCTION OF TOXICITY, MOBILITY, VOLUME AND PERSISTENCE THROUGH TREATMENT

Each alternative, with the exception of the No Action Alternative, requires varying degrees of contaminated soil removal and placement of a "clean" fill cover to create a barrier between underlying soil contaminants and the residential population. Alternative 8 is the only alternative to incorporate treatment as part of the remedial action. This treatment would solidify the excavated soil and would likely reduce the metals mobility from soils at the disposal area. The additional decrease in mobility by pozzolanic treatment is not known.

All alternatives would increase volume of soil remaining within the Superfund boundaries through bulking (10 to 15 percent of the in-place volume). The volume would increase by approximately 50 percent as a result of the pozzolanic treatment in Alternative 8 as compared to Alternative 3. None of the alternatives proposes to change the toxicity or persistence of the contaminants.

8.5 SHORT-TERM EFFECTIVENESS

Most of the remedial actions are similar in the technologies proposed for implementation. The extent of the remedial action varies considerably among alternatives. Alternatives 3, 5, and 8 are generally equivalent in the amount of short-term risk they pose to the community. Each requires the removal of the top vegetative layer and varying amounts of underlying soil. Each alternative would include continuing to prioritize residential yards on the basis of sensitive subpopulations. Completion of these alternatives would require 4 to 6 years. Alternative 6 would require considerably more time to complete because of its soil removal requirements. Exposure to fugitive dust generated by the remedial activities is the common risk shared by each alternative. Localized releases of metals-laden dust would likely occur during excavation, but such releases would be minimized by dust control techniques. However, none of the action alternatives is expected to substantially affect the communities during remediation.

Alternative 6 would create a slightly higher risk to workers and residents than the other alternatives, mainly because of the volumes of materials to be excavated and moved and the duration of time needed to accomplish Alternative 6. The greater excavation volume would be associated with increased noise and greater annoyance of residents from more construction activity. Heavy equipment traffic would also increase on local roads with implementation of Alternative 6.

Construction contractors would need protection against dermal and respiratory exposure to the dust while working in contaminated areas. Protective clothing and respirators or dust masks would help control this risk. These risks are inherent to all alternatives.

8.6 IMPLEMENTABILITY, RELIABILITY, AND CONSTRUCTIBILITY

In general, there is not a great difference among alternatives in the types of remedial activities proposed. The extent or degree to which the remediation is applied does vary significantly between alternatives. Most of the activities proposed as part of the alternatives

including disposal are well-developed technologies. All of these activities are technically feasible, but the level of effort associated with each is different.

Alternative 5 is the most easily implemented alternative proposed, requiring only the removal and replacement of a sod and grass layer. However Alternative 5 was judged to be the least reliable because of lack of durability and difficulty in implementing and enforcing the extensive associated institutional controls requirements. Alternative 6, however, is the most difficult to construct, requiring removal of up to 7 feet of soil around each residence, and resulting in potential complications associated with exposed structure footings, utility lines, and pipes. Because of this, Alternative 6 has the greatest potential to impact the community through construction delays resulting from complications. Alternatives 3 and 8 are implementable, reliable, and constructible and require slightly more complex activities than Alternative 5, involving the removal of up to 12 inches of soil and the vegetation layer with subsequent replacement of at least 12 inches of "clean" soil and a new sod layer.

8.7 COST

The cost comparisons are straightforward. Comparing present worth costs, Alternative 6 is the most expensive and Alternative 5 is the least expensive of the action alternatives. The costs of the action alternatives, including present worth, are listed in Table 8-1.

Table 8-1 Summary of Estimated Costs
(See Data in Original Document)

8.8 STATE ACCEPTANCE

This decision document presents the remedial action selected by the U.S. EPA and IDHW for the Populated Areas Residential Soils Operable Unit at the Bunker Hill Mining and Metallurgical Complex

8.9 COMMUNITY ACCEPTANCE

U.S. EPA and IDHW solicited input from the community on the methods proposed for residential soils. Public comments, in general, indicated support for the recommendation of Alternative 3 in the proposed plan and urged an expeditious implementation of the plan. Public comments are specifically addressed in the Responsiveness Summary section of this document and some have been incorporated into the selected remedy.

9 THE SELECTED REMEDY

9.1 INTRODUCTION

IDHW and U.S. EPA have selected Alternative 3 (as modified by public comments) as the remedy for contaminated residential soils at the Bunker Hill site. This selection

For each residential yard, the exact nature of the remediation (i.e., how much sod to replace, which bushes to remove, etc.) would have to be considered on a case-by-case basis. However, for consistency, the following areas would generally be remediated within each yard:

- o Sod areas
- o Roadway shoulders (if curb and gutter are not present) to asphalt or pavement and to the lateral extension of property lines
- o Alleys (if unpaved) to the extension of the lot lines
- o Landscaped areas
- o Garden areas
- o Unpaved driveways
- o Garages with dirt floors
- o Storage areas

Areas immediately associated with the residential properties (i.e., road shoulders and alleys) will not require top soil, but will require replacement will clean material in kind or a permanent cover. Any steep hillside areas located immediately adjacent to yards and with a soil lead concentration greater than the threshold level will be stabilized as part of this action to prevent runoff and recontamination. The final remedy for the hillsides will be addressed in a subsequent ROD.

Based on dose response modeling, a threshold level of 1,000 ppm lead in residential soil was determined to be the threshold cleanup level most appropriate for this site. The result of the threshold assessment, and the assumptions used, are summarized in Table 9-1.

Table 9-1 Risk Range for a Threshold Level of 1,000 ppm
(See Data in Original Document)

Requirements for removal and replacement of soils on areas adjacent to residential lots, such as vacant residential lots, within the Populated Areas will be the same as for occupied properties.

VISUAL MARKER

For residential yards that require excavation to 12 inches, if the results of sampling in the 12- to 18-inch interval exceed the threshold level, a visual marker (such as erosion control fabric or other suitable material) will be placed prior to backfilling with clean

fill.

REVEGETATION

During the excavation process, all existing sod and soil coverings will be removed and disposed of along with the soil. Larger trees and shrubs will be left in place but subject to pruning. After spreading compaction, and grading, clean fill will be revegetated. The lawn areas of remediated yards will generally be revegetated with sod. Steep hillsides and other remediated areas not currently planted with lawns (such as vacant lots) will be stabilized and hydroseeded with native grasses. If preferred by a property owner, hydroseeding with native grasses could be substituted for the sod. Vacant lots will be hydroseeded with native grasses after remediation. To the extent practicable, all yard landscaping will be returned to its original condition.

DUST SUPPRESSION

Dust suppression measures will be implemented throughout the remediation process to reduce exposure of workers and residents to airborne contaminants. Dust suppression will include, but not be limited to:

- o Watering of residential yard areas prior to excavation activities
- o Continued watering during excavating, as necessary
- o Placement of tarps or covers over excavated materials
- o Use of tarps or covers over truck beds to reduce blowing dust and spillage during transportation to the waste repository
- o Daily cleanup of all spilled or tracked soils from sidewalks, roadways, etc.

DISPOSAL OF CONTAMINATED MATERIALS

The analysis of Applicable or Relevant and Appropriate Requirements associated with the disposal of contaminated residential soils assumed that the soils repository would be located within the Bunker Hill site. It is recommended that Page Ponds be used for the disposal repository because it has adequate volume, is within the Bunker Hill site, and the action will reduce the contaminated windblown dust originating from the Page Ponds area.

The use of Page Ponds as the repository will require that it be capped to minimize airborne contaminant migration and reduce the threat of direct contact exposure. The cap surface area will be compacted and graded to prevent ponding and minimize infiltration; it will also be vegetated for stabilization and moisture absorption. Access to the area will be restricted by fencing, locked gates, and warning signs. Future use of the repository will be limited and subject

to institutional controls.

If Page Ponds is not used as the residential soil repository, the chosen repository site will be subject to agency evaluation and public notification.

INSTITUTIONAL CONTROLS

The goal of the institutional controls program is to develop a flexible system that builds on existing administrative structures and programs rather than create a new layer of bureaucracy. Institutional controls regulation will be uniform throughout the Bunker Hill site, irrespective of jurisdictional boundaries. The institutional controls associated with this ROD are designed for the maintenance of residential soil barriers only. These controls are necessary and are an integral part of the selected remedy.

Physical Program Requirements

Planning Zoning, Subdivision and Building Permit Regulations:

Implementation of planning, zoning and subdivision controls through local ordinances, designed to protect and maintain barriers when development of any action that would breach a barrier takes place.

Disposal of Unearthed Contaminants:

When a barrier is broken, contaminated soils that are removed must be handled to minimize exposure, collected for disposal, and transported to a proper disposal site. A means for disposal of incidental contaminated soils will be provided to residents.

Provision of Clean Soil:

A program will be implemented to provide a centrally located supply of clean replacement soil (both fill to topsoil) to facilitate barrier repair, maintenance, and establishment of produce garden areas.

Administrative Program Requirements

Coordination of Public Institutions:

Effective administration of a uniform Institutional Controls Program will require shared authority and resources. The four cities and Shoshone County will play an important role through already established permitting procedures. It has been recommended that the Panhandle Health District will administer the effort with permitting, inspection, records maintenance, and enactment of regulations, where necessary, across jurisdictional boundaries.

Deed Notices:

These are a method to notify new owners of their responsibility for participation in that system.

Educational Programs:

Educational programs will be developed to keep information about the barrier system in the public eye and to help the public recognize when disruption of the barrier systems requires attention or caution. Distribution of information should be provided through pamphleting, brochures, and general media exposure.

Permitting and Inspection Procedures:

Permit issuance and recordkeeping procedures should be tailored to minimize inconvenience to permit applicants. A permit system that integrates with existing permit routines will be implemented.

Monitoring and Health Surveillance Programs:

Monitoring will be required to assure both program performance and effectiveness. Health intervention efforts will be required to document and assess success in achieving remedial goals and objectives.

An Evaluation of Institutional Controls for the Populated Areas of the Bunker Hill Superfund Site outlines the various options associated with each of the institutional control requirements and will be used in the remedial design phase to guide implementation of the program. The implementation phase, referred to as Phase II, will include passing local ordinances, setting up an administrative system to oversee and run the program, and documentation of detailed procedures for each of the program components.

MONITORING

The effectiveness of the institutional controls programs will be evaluated periodically. Appropriate air monitoring will be conducted to identify the occurrence of contaminant migration during remedial activities. Any exceedances of the standards will result in immediate implementation of additional dust suppression measures or a shutdown of construction activities.

Since contaminated material will be left onsite, both in Populated and Non-populated Areas, ongoing monitoring of fugitive dust and residential yards is necessary to ensure that the clean barrier is maintained.

9.3 CHANGES TO PROPOSED PLAN

During the public comment period, several issues were raised concerning the preferred alternative in the Proposed Plan; consequently, several minor modifications have been incorporated into the selected remedy in response to those concerns. The following is a list of those modifications:

o Depth of excavation may be variable (less than 12 inches) depending on depth of contamination.

o For those properties requiring a visual marker, it will be a material that can be easily seen during digging or excavation activities. The visual marker does not have to be a 2-inch gravel layer.

o Requirements for disposal site closure included an impermeable cap to protect groundwater. ARARs associated with groundwater and surface water protection will be addressed in a subsequent FS and ROD.

o The scope of the institutional controls program will be reevaluated periodically because the requirements of a program of this nature may change with time.

o Soil will be provided for homeowners who have a soil lead level less than 1,000 but who want a garden.

9.4 COST

Cost evaluations, including the assumptions used, are presented in the Feasibility Study. A summary of the capital costs associated with the selected alternative is shown in Table 9-2. The costs are order-of-magnitude (+50 percent to -30 percent) estimates. Capital costs are those required to initiate and construct the remedial action. Typical capital costs include construction equipment, labor and materials expenditures, engineering, and construction management. Bid and scope contingencies are also included in the total capital cost. Projected annual operation and maintenance costs for the selected remedy are also presented in Table 9-2. These costs are necessary to ensure the continued effectiveness of a remedial action. Included are such items as labor and materials; monitoring and the institutional controls program; and insurance, taxes, etc.

Table 9-2 Summary of Estimated Costs for Selected Remedy
(See Data in Original Document)

The feasibility level cost estimates shown have been prepared for guidance in project evaluation and implementation from the information available at the time of the estimate. The final costs of the project will depend on actual labor and material costs, actual site conditions, productivity, competitive market conditions, final project scope and schedule, and other variable factors. As a result, the final project costs will vary from the estimates presented here.

Present worth costs are calculated using a 5 percent discount rate and a 30-year estimated project life. The present worth cost for the selected remedy is $40.6 million (Table 9-2). Capital costs and long-term annual operations and maintenance (O&M) costs are included in the total present worth cost. Longterm O&M costs are those associated with maintaining an alternative after implementation is complete.

Costs presented in Table 9-2 are lower than those presented in the Residential Soil Feasibility Study or the Proposed Plan. The reduction in cost is associated with changes to the Proposed Plan as presented in Section 9.3. Specially, removing the requirement for an impermeable cap accounts for the cost reduction.

9.5 PERFORMANCE REQUIREMENTS

A remedial objective for this operable unit is to decrease the exposure to lead-contaminated residential soils such that 95 percent have blood leads greater than 15 ug/dl. The former is projected to be achieved by reducing the overall soil and dust loading concentration to 700 to 1,200 ppm. The 1,000 ppm yard soil threshold cleanup level will mean yard soil concentrations to approximately 200 to 300 ppm in residential areas. In combination with other remedial measures and the positive effects likely to be seen in other media, it is expected that this objective will be met. Achieving the latter objective of less than 1 percent of area children with blood lead concentrations below 15 ug/dl is less dependent on the mean soil/dust concentrations than on the soil concentration left in an unremediated yard. A child living on an unremediated yard of 1,000 ppm is estimated to have a 0.1 to 2.5 percent (depending on various assumptions) chance of exceeding 15 ug/dl blood lead in the Bunker Hill post-remediation environment. Any higher threshold cleanup level would result in unacceptable risk to that child. It is expected that this goal will be achieved by replacing all residential yards with a lead concentration greater than 1,000 ppm lead with clean material (less than 100 ppm). This expectation assumes that fugitive dust sources will be controlled and house dust concentrations will consequently decrease and that remediated yards will not be recontaminated.

This remedy mitigated the risks associated with the following pathways identified in the risk assessment:

- Inhalation/Ingestion of Contaminated Residential Soil
- Ingestion of Locally Grown Produce

This remedy does not directly address the risks associated with the following pathways identified in the risk assessment:

- Consumption of Contaminated Groundwater
- Inhalation/Ingestion of Windblown Dust
- Inhalation/Ingestion of Contaminated House Dust

Actions are being taken now to address these risks. The final remediation with respect to these risks will be addressed in a subsequent feasibility study.

10 STATUTORY DETERMINATIONS

The selected remedy for residential soils is protective of human health and the environment, will comply with federal and state requirements that are legally applicable or relevant and appropriate, and is cost-effective. The selected remedy does utilize alternative treatment and resource recovery technologies to the maximum extent practicable. However, since no treatment and resource recovery technologies were found to be practicable, none were incorporated into the remedy. Because this remedy will result in hazardous substances remaining onsite above health-based levels, the 5-year review provisions of CERCLA Section 121c will apply to this action. The following sections discuss how the selected remedy meets the statutory requirements.

10.1 PROTECTION OF HUMAN HEALTH AND THE ENVIRONMENT

Lead absorption among young children is the most significant health risk posed by this site. Residential soils were identified in the RADER to be one of the primary contributors to risk associated with sub-chronic lead absorption. In order to reduce blood lead exposures, the selected remedy replaces metal-contaminated residential soils with uncontaminated soil, thereby breaking the exposure pathway between soils and children. Post-remediation modeling scenarios show the soil cleanup level of 1,000 ppm will result in a sitewide mean blood lead level of 2.7 to 3.9 ug/dl. Only 1 to 3 percent of the children living onsite are predicted to have blood lead levels in excess of 15 ug/dl. It is expected that at least 95 percent will have a blood lead level less than 10 ug/dl.

Inclusion of produce garden area remediation to a depth of 24 inches will also reduce the exposure to cadmium, lead, and zinc associated with consumption of local garden produce.

The remedy selection will also effectively mitigate chronic noncarcinogenic risks associated with ingestion of antimony, cadmium, and mercury via soil ingestion. Carcinogenic risks associated with arsenic and cadmium exposure through fugitive dust will be addressed under a separate operable unit.

Contaminated residential soils will be consolidated in a permanent repository. All consolidation areas will be protected from erosion and surface infiltration by a revegetated topsoil cap and contouring. Experience with residential soil removal actions during 1989 and 1990 indicate that with appropriate precautions there will be no unacceptable short-term risks or cross-media impacts associated with the implementation of the selected remedy.

The institutional controls program will ensure the maintenance of physical and institutional barriers that protect against metal exposure. Continued blood lead and residential soils monitoring will measure the long-term success of the selected remedy.

House dust has also been identified as a significant lead exposure pathway. Residential soils are a contaminant source to house dust. Thus, remediating residential soils will reduce a contamination pathway to home interior. Fugitive dust will need to be controlled and monitored concomitant with residential soil remediation to minimize soil recontamination. The RADER discusses the rate of soil recontamination from airborne fugitive dust and recommends that airborne dust be reduced substantially. Control of fugitive dust will also eliminate direct exposure to highly concentrated dusts, reduce accumulation of metals in homes, and prevent excessive deposition on homegrown produce in local gardens. Dust control measures have been taken on the site in the past 2 years. These measures include irrigation of the Central Impoundment Area (CIA), revegetation of some of the Bureau of Land Management (BLM) property on Smelterville Flats, placement of large rocks on barren areas north of the Kellogg Middle School, and spreading of sawdust on the Smelterville Flats area. Control of fugitive dust from barren hillsides is being addressed in the hillside revegetation order previously discussed. Additional dust control measures will be implemented by the potentially responsible parties (PRPs) under the July 1991 Administrative Order on Consent (see Section 2.5).

The analysis presented in the RADER and the FS shows that the remedy selected for residential soils will break the significant exposure pathways associated with soil. Once residential soil removal is completed, waste soils will be consolidated within the area of contamination of the Bunker Hill site, and an institutional controls program is implemented, risks associated with metal-contaminated residential soils will be mitigated. Therefore, IDHW and U.S. EPA have concluded that the selected remedy for residential soils will be protective of public health and the environment.

10.2 COMPLIANCE WITH APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARs)

Pursuant to SARA Section 121(d), remedial actions shall attain a degree of cleanup of hazardous substances, pollutants, and contaminants released into the environment and control of further release which, at a minimum, assures protection of human health and the environment. In addition, remedial actions shall, upon their completion, reach a level or standard of control for such hazardous substances, pollutants, or contaminants which at least attains legally applicable or relevant and appropriate federal standards, requirements, criteria, or limitations, or any promulgated standards, requirements, criteria, or limitations under a state environmental or facility siting law that is more stringent than any federal standard (ARARs). All ARARs would be met by the selected remedy.

The federal and state ARARs identified by U.S. EPA and IDHW, respectively, for residential soil removal are presented in Tables 10-1 through 10-6. An evaluation of chemical, location, and action-specific ARARs is presented in Section 2 of the Residential Soils Focused Feasibility Study. Additional discussion of chemical-specific ARARs and other requirements to be considered (TBCs) is presented in Section 3 of the RADER.

Table 10-1 Federal Chemical-Specific ARARs
(See Data in Original Document)

Table 10-2 Federal Location-Specific ARARs
(See Data in Original Document)

Table 10-3 Federal Action-Specific ARARs
(See Data in Original Document)

Table 10-4 State of Idaho Chemical-Specific ARARs
(See Data in Original Document)

Table 10-5 State of Idaho Location-Specific ARARs
(See Data in Original Document)

Table 10-6 State of Idaho Action-Specific ARARs
(See Data in Original Document)

There are currently no promulgated laws or standards for lead in soil. However, a site-specific threshold level of 1,000 ppm lead in residential soil, that is expected to result in a community average of 200 to 300 ppm, has been developed for protection of human health.

For the Bunker Hill residential soils action, contaminated residential soil will be consolidated from yards throughout the site into a single location. Since some residential soils did demonstrate RCRA hazardous characteristics for lead and pesticides (chlordane), an analysis of the applicability or relevance and appropriateness of the RCRA hazardous waste regulations is required:

For RCRA to be applicable, the material must demonstrate hazardous characteristics, and the proposed action must involve either treatment, storage, or disposal of the material as defined by RCRA. As the Remedial Investigation sampling and analysis has shown, residential properties and all other areas within the Bunker Hill Superfund Site are contaminated to various degrees with lead and other heavy metals. Contamination is contiguous throughout the site and the site is considered a single "area of contamination" (AOC). As described in the preamble to the final NCP, movement of wastes and soil within an AOC at a Superfund site does not constitute disposal or "placement" and therefore does not trigger RCRA, Subtitle C, disposal requirements. For this action, all soil consolidation and movement will be within a single AOC; thus, the RCRA requirements are not applicable.

For RCRA to be relevant and appropriate, the RCRA requirements must address problems or situations that are similar to the action being taken and the requirements must be well suited to the site. U.S. EPA has determined that portions of the RCRA closure requirement are relevant and appropriate for this action.

Closure requirements address what actions are necessary to protect public health and the environment when the disposal action is complete. For this action, the relevant and appropriate closure requirements include: 1) capping to minimize airborne contaminant migration and reduce the threat of direct contact exposure; 2) long-term management of the disposal site, including cover maintenance and groundwater monitoring; and 3) institutional controls such as access restrictions, land use restrictions, and/or deed notices.

Closure requirements and landfill design and operating requirements with respect to groundwater and surface water protection will be addressed in a subsequent ROD.

RCRA minimum technology requirements are not appropriate for this action because the residential soils do not present hazards that warrant secure disposal.

Requirements of the Land Disposal Restrictions are not appropriate for this remedial action because the material will be removed within the AOC. Placement, as defined by RCRA, will not occur.

If Page Ponds is not used as the residential soils repository, the agencies will conduct an evaluation of ARARs specific to the repository site chosen.

IDHW and U.S. EPA have determined that all state and federal ARARs for residential soils removal and replacement will be met by the selected remedy. The agencies have not determined the ARARs with respect to groundwater and surface water protection as part of this operable unit ROD. That determination will be made in a subsequent ROD.

10.3 COST-EFFECTIVENESS

IDHW and U.S. EPA believe the selected remedy is cost-effective in mitigating the risk posed by contaminated residential soils. Section 300.430(f)(ii)(D) of the National Contingency Plan (NCP) requires an evaluation of cost-effectiveness by comparing all the alternatives that meet the threshold criteria (protection of human health and the environment) against three additional balancing criteria (long-term effectiveness and permanence; reduction of toxicity, mobility, or volume through treatment; and short-term effectiveness). The selected remedy meets these criteria and provides overall effectiveness in proportion to its cost.

The selected remedy includes removing and replacing contaminated soils (or placing a soil cap, where appropriate), installing visual barriers (where applicable), revegetating, suppressing dust during remediation, disposing of contaminated materials, and monitoring for metals in soil. Institutional controls will ensure long-term maintenance of physical and institutional barriers that protect against metals exposure. This alternative is attractive because of the relatively low cost (approximately $41.3 million present worth) and expected effectiveness, as compared with other alternatives.

The principal difference between the selected remedy and two of the other alternatives is excavation depth. One alternative involves sod excavation and replacement without removal of underlying contaminated soils. Although less expensive than the selected remedy, sod

removal and replacement would provide a less effective means of protecting human health and the environment. Another alternative, which required a 7-foot excavation depth, was considered excessive. Although an excavation depth of 7 feet would effectively remove the contaminated residential soils, the associated cost of $193 million was substantially higher than that for the selected remedy. The added remedial effectiveness would be marginal with respect to the additional cost.

An alternative with a pozzolanic treatment prior to disposal was also evaluated. Pozzolanic treatment would be intended to reduce the mobility of contaminants, as compared with untreated contaminated soil. However, the reduction in contaminant mobility is expected to be marginal with respect to the additional cost of $14.7 million. Contaminants in untreated soils would be adequately immobilized when disposed in a revegetated and properly contoured landfill. The selected alternative was therefore determined to be more cost-effective.

10.4 UTILIZATION OF PERMANENT SOLUTIONS AND ALTERNATIVE TREATMENT TECHNOLOGIES TO THE MAXIMUM EXTENT PRACTICABLE

IDHW and U.S. EPA believe the selected remedy represents the maximum extent to which permanent solutions and treatment technologies can be utilized in a cost-effective manner for residential soils at the Bunker Hill site. Of the alternatives protective of human health and the environment and that comply with ARARs, the selected remedy provides the best balance in terms of long-term effectiveness and permanence; reduction of toxicity, mobility, volume, and persistence; short-term effectiveness; implementability; and cost. Also, the selected remedy considers the statutory preference for treatment as a principal element and considers community acceptance.

Long-term effectiveness was the primary reason for selecting Alternative 3 over Alternative 5. Twelve inches of soil and sod provide a much more permanent physical barrier to potential exposure than simply a sod barrier. The institutional controls associated with Alternative 3 improved community acceptance because the controls are less intrusive compared to Alternative 5. The cost of removing soils to a depth of 7 feet in Alternative 6 was too high compared to Alternative 3, considering the associated incremental improvement in permanence.

The selected remedy does utilize alternative treatment and resource recovery technologies to the maximum extent practicable. Treatment of residential soils was not found to be practicable; therefore, this remedy does not satisfy the statutory preference for treatment as a principal element. The combination of high soil volume, the nature of metal contamination, and the need to excavate soils from yards prior to application of a treatment technology like soil washing made the costs of any known treatment technology, whether proven or unproven, prohibitive. An in situ soil treatment process would have eliminated the soil handling requirement. However, fixation or pozzolanic treatments are not consistent with the uses of a residential yard. There are no other in situ treatment technologies known to be effective in removing metals from soil.

10.5 PREFERENCE FOR TREATMENT AS A PRINCIPAL ELEMENT

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

JUL 1 1994

OFFICE OF SOLID WASTE AND EMERGENCY RESPONSE

OSWER Directive # 9355.4-12

MEMORANDUM

SUBJECT: Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities

FROM: Elliott P. Laws
Assistant Administrator

TO: Regional Administrators I-X

PURPOSE

As part of the Superfund Administrative Improvements Initiative, this interim directive establishes a streamlined approach for determining protective levels for lead in soil at CERCLA sites and RCRA facilities that are subject to corrective action under RCRA section 3004(u) or 3008(h) as follows:

- It recommends screening levels for lead in soil for residential land use (400 ppm);[1]
- It describes how to develop site-specific preliminary remediation goals (PRGs) at CERCLA sites and media cleanup standards (MCSs) at RCRA Corrective Action facilities for residential land use; and,
- It describes a plan for soil lead cleanup at CERCLA sites and RCRA Corrective Action facilities that have multiple sources of lead.

This interim directive replaces all previous directives on soil lead cleanup for CERCLA and RCRA programs (see the Background section, 1989-1991).

KEY MESSAGES

Screening levels are not cleanup goals. Rather, these screening levels may be used as a tool to determine which sites

[1] The residential screening level is the same concept as the action level proposed in the RCRA Corrective Action Subpart S rule (July 27, 1990, 55 *Federal Register* 30798).

or portions of sites do not require further study and to encourage voluntary cleanup. Screening levels are defined as a level of contamination above which there may be enough concern to warrant site-specific study of risks. Levels of contamination above the screening level would NOT automatically require a removal action, nor designate a site as "contaminated."

The residential screening level for lead described in this directive has been calculated with the Agency's new Integrated Exposure Uptake Biokinetic Model (IEUBK) model (Pub. # 9285.7-15-2, PB93-963511), using default parameters. As outlined in the Guidance Manual for the IEUBK Model for Lead in Children (Pub. # 9285.7-15-1, PB93-963510, February 1994), this model was developed to: recognize the multimedia nature of lead exposures; incorporate important absorption and pharmacokinetic information; and allow the risk manager to consider the potential distributions of exposure and risk likely to occur at a site (the model goes beyond providing a single point estimate output). For these reasons, this approach is judged to be superior to the more common method for assessing risks of non-cancer health effects which utilizes the reference dose (RfD) methodology. Both the Guidance Manual and the model are available to Superfund staff through the Superfund Document Center (703-603-8917) and to the public through the National Technical Information Service (703-487-4650).

Residential preliminary remediation goals (PRGs) for CERCLA remediations and media cleanup standards (MCSs) for RCRA corrective actions can be developed using the IEUBK model on a site-specific basis, where site data support modification of model default parameters. At some Superfund sites, using the IEUBK model with site-specific soil and dust characteristics, PRGs of more than twice the screening level have been identified. However, it is important to note that the model alone does not determine the cleanup levels required at a site. After considering other factors such as costs of remedial options, reliability of institutional controls, technical feasibility, and/or community acceptance, still higher cleanup levels may be selected.

The implementation of this guidance is expected to provide for more consistent decisions across the country and improve the use of site-specific information for RCRA and CERCLA sites contaminated with lead. The implementation of this guidance will aid in determining when evaluation with the IEUBK model is appropriate and in assessing the likelihood that environmental lead poses a threat to the public. Use of the IEUBK model in the context of this guidance will allow risk managers to assess the

contribution of different environmental sources of lead to overall blood lead levels (e.g., consideration of the importance of soil lead levels relative to lead from drinking water, paint and household dust). It offers a flexible approach to considering risk reduction options (referred to as the "bubble" concept) that allows for remediation of lead sources that contribute significantly to elevated blood lead. This guidance encourages the risk manager to select, on a site-specific basis, the most appropriate combination of remedial measures needed to address site-specific lead exposure threats. These remedial measures may range widely from intervention to abatement. However, RCRA and CERCLA have very limited authority to address interior exposures from interior paint. For a detailed discussion of the decision logic for addressing lead-contaminated sites, see the Implementation section and Appendix A.

Relationship to lead paint guidance. In addition, this interim directive clarifies the relationship between guidance on Superfund and RCRA Corrective Action cleanups, and EPA's guidance on lead-based paint hazards (discussed further in Appendix C). The paint hazard guidance will be issued to provide information until the Agency issues regulations identifying lead-based paint hazards as directed by Section 403 of the Toxic Substances Control Act (TSCA)[1]. Lead-based paint hazards are those lead levels and conditions of paint, and residential soil and dust that would result in adverse health effects.

The two guidance documents have different purposes and are intended to serve very different audiences. As a result the approaches taken differ to some degree. The lead-based paint hazard guidance is intended for use by any person who may be involved in addressing residential lead exposures (from paint, dust or soil.) It thus relates to a potentially huge number of sites, and serves a very broad potential audience, including private property owners or residents in addition to federal or state regulators. Much residential lead abatement may take place outside any governmental program, and may not involve extensive site-specific study.

This OSWER guidance, on the other hand, deals with a much smaller number of sites, being addressed under close federal regulatory scrutiny, at which extensive site characterization will have been performed before cleanup decisions are made. Thus, the RCRA and CERCLA programs will often have the benefit of much site-specific exposure information. This guidance is intended for use by the relatively small number of agency officials who oversee and direct these cleanups.

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[1]Title IV of TSCA (including section 403) was added by the Residential Lead-Based Paint Hazard Reduction Act of 1992 (Title X of the Housing and Community Development Act of 1992).

Both the TSCA Section 403 and OSWER programs use a flexible, tiered approach. The OSWER guidance sets a residential screening level at 400 ppm. As noted above, this is not intended to be a "cleanup level" for CERCLA and RCRA facilities, but only to serve as an indicator that further study is appropriate. The Section 403 guidance indicates that physical exposure-reduction activities may be appropriate at 400 ppm, depending upon site-specific conditions such as use patterns, populations at risk and other factors. Although worded somewhat differently, the guidances are intended to be similar in effect. For neither guidance is 400 ppm to automatically be considered a "cleanup level"; instead, it indicates a need for considering further action, but not necessarily for taking action. Neither is meant to indicate that cleanup is necessarily appropriate at 400 ppm. The greater emphasis in this OSWER guidance on determining the scope of further study reflects the fact that both CERCLA and RCRA cleanups proceed in stages with detailed site characterization preceding response actions in every case.

Above the 400 ppm level, the Section 403 guidance identifies ranges over which various types of responses are appropriate, commensurate with the level of potential risk reduction, and cost incurred to achieve such risk reduction. For example, in the range of 400 to 5000 ppm, limited interim controls are recommended depending, as noted above, on conditions at the site, while above 5000 ppm, soil abatement is recommended. This OSWER guidance does not include comparable numbers above 400 ppm; instead, as discussed above, it recommends the site-specific use of the IEUBK model to set PRGs and MCSs, when necessary. The remedy selection process specified in the National Contingency Plan (NCP) should then be used to decide what type of action is appropriate to achieve those goals.

In general, because the Section 403 guidance was developed for a different purpose and audience, OSWER does not recommend that it be used as a reference in setting PRGs and MCSs or in determining whether action at a particular site is warranted. (To put it another way, it generally should not be treated as a "to be considered" document or "TBC" under CERCLA.) The section 403 guidance is meant to provide generic levels that can be used at thousands of widely varying sites across the nation. The detailed study that goes on at CERCLA or RCRA sites will allow levels to be developed that are more narrowly tailored to the individual site. Nothing in the section 403 guidance discourages setting more site-specific levels for certain situations; in fact, it specifically identifies factors such as bioavailability that may significantly affect the evaluation of risk at some sites.

The IEUBK model. The Agency is further studying both the IEUBK model and analyses of epidemiologic studies in order to better develop the technical basis for rulemaking under TSCA

Section 403. The Agency intends to promulgate regulations under Section 403 setting health-based standards for lead in soil and dust. OSWER intends to issue a final soil lead directive once the TSCA Section 403 regulations are finalized. For additional information on TSCA Section 403 developments, call (202) 260-1866.

However, the Agency believes that risk managers (risk assessors, on-scene coordinators, remedial project managers, and other decision-makers at Superfund and RCRA sites) are currently in need of the best guidance available today. The Agency believes that the IEUBK model is the best available tool currently available for assessing blood lead levels in children. Furthermore, use of the IEUBK provides allows the risk manager to consider site-specific information that can be very important in evaluating remediation options. Therefore, using the latest developments in the IEUBK model and the collective experience of the Superfund, RCRA Corrective Action, and TSCA Section 403 programs, the Agency is offering this guidance and is recommending a residential screening level for Superfund and RCRA sites of 400 ppm.

BACKGROUND

Early OSWER guidance (1989-1991). Four guidance documents on soil lead cleanup were issued by OSWER during the period of 1989 to 1991:

1. September 1989, OSWER Directive #9355.4-02. This guidance recommended a soil lead cleanup level of 500 - 1000 ppm for protection of human health at residential CERCLA sites.

2. May 9, 1990. RCRA Corrective Action program guidance on soil lead cleanup. This guidance described three alternative methods for setting "cleanup levels" (not action levels) for lead in soil at RCRA facilities. One approach was to use levels derived from preliminary results of IEUBK model runs. The other two approaches were to use the range of 500 to 1000 provided in the 1989 directive on CERCLA sites, or to use "background" levels at the facility in question.

3. June 1990, OSWER Directive #9355.4-02A. Supplement to Interim Guidance on Establishing Soil Lead Cleanup Levels at Superfund Sites. This memorandum reiterated that the September 1989 directive was guidance and should not be interpreted as regulation.

4. August 29, 1991. This supplemental guidance discussed EPA's efforts to develop a new directive that would

accomplish two objectives: (1) account for the contribution from multiple media to total lead exposure; and, (2) provide a stronger scientific basis for determining a soil lead cleanup level at a specific site.

Development of the IEUBK Model for OSWER use. During the 1989-91 time period, use of the EPA IEUBK model was identified as the best available approach for accomplishing the objectives outlined in the August 1991 guidance. The model integrates exposure from lead in air, water, soil, dust, diet, and paint with pharmacokinetic modeling to predict blood lead levels in children (i.e., children 6 to 84 months old), a particularly sensitive population.

In the spring of 1991, OSWER organized the Lead Technical Review Workgroup to assist Regional risk assessors and site managers in both using the model and making data collection decisions at CERCLA and RCRA sites. The workgroup was composed of scientists and risk assessors from the Regions and Headquarters, including the Office of Research and Development (ORD), and the Office of Pollution Prevention and Toxic Substances (OPPTS).

In November 1991, the EPA Science Advisory Board (SAB) reviewed the scientific merits of using the IEUBK model for assessing total lead exposure and developing soil lead cleanup levels at CERCLA and RCRA sites. In general, the SAB found the model to be an important advance in assessing potential health risks from environmental contaminants. However, the SAB also recommended additional guidance on the proper use of the model.

In response to SAB concern over the potential for incorrect use of the model and selection of inappropriate input values both for default and site-specific applications, OSWER developed a comprehensive "Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children" (referred to in this interim directive as the "Guidance Manual"). This Guidance Manual assists the user in providing inputs to the model to estimate risks from exposures to lead. It discusses the use of model default values or alternative values, and the application of the model to characterize site risks. Use of the Guidance Manual should facilitate consistent use of the IEUBK model and allow the risk assessor to obtain valid and reliable predictions of lead exposure. The Lead Technical Review Workgroup has been collecting data to further validate the model and to update the Guidance Manual as needed.

Relationship to RCRA Corrective Action "Action" Levels. The approach for calculating a screening level for lead (including exposure assumptions), set forth in this Revised Interim Soil Lead Directive, supersedes the guidance provided for calculating

"action" levels set forth in Appendix D of the proposed Subpart S Corrective Action rule. In the July 27, 1990 RCRA proposal (55 Federal Register 30798), EPA introduced the concept of "action levels" as trigger levels for further study and subsequent remediation at RCRA facilities. In this respect, the current directive's "screening levels" are analogous to the proposed rule's "action levels." In the proposal, where data were available, action levels were developed for three pathways of human exposure to contaminants: soil ingestion, water ingestion and inhalation of contaminated air. Exposure assumptions used in the calculations were set out in Appendix D of the proposal. For the soil pathway, action levels were calculated two different ways depending on whether the contaminant in the soil was a carcinogen or a systemic toxicant. Although lead was listed in Appendix A of the preamble to the rule as a class B2 carcinogen, no action level had been calculated because neither a carcinogenic slope factor (SF) nor a reference dose (RfD) had been developed by the Agency. Although the guidance in Appendix D of the proposed Corrective Action rule remains in effect with respect to other hazardous constituents, this directive now allows for the development of the lead screening ("action") level using the IEUBK model.

Recent developments (1992-Present). Following discussions among senior Regional and OSWER management, the OSWER Soil Lead Directive Workgroup (composed of Headquarters, Regional and other Federal agency representatives) recommended in the spring of 1992 that a "two step" decision framework be developed for establishing cleanup levels at sites with lead-contaminated soils. This framework would identify a single level of lead in soils that could be used as either the PRG for CERCLA site cleanups or the action level for RCRA Corrective Action sites, but would also allow site managers to establish site-specific cleanup levels (where appropriate) based on site-specific circumstances. The IEUBK model would be an integral part of this framework. OSWER then developed a draft of this directive which it circulated for review on June 4, 1992. The draft set 500 ppm as a PRG and an action level for RCRA facilities in residential settings.

Following development of this draft, OSWER held a meeting on July 31, 1992 to solicit a broad range of views and expertise. A wide range of interests, including environmental groups, citizens and representatives from the lead industry attended. This meeting encouraged OSWER to think more broadly about how the directive would affect urban areas, how lead paint and dust contribute to overall risk, and how blood lead data could be used to assess risk. In subsequent meetings with the Agency for Toxic Substances and Disease Control (ATSDR) and the Centers for Disease Control (CDC), options were discussed on how to use blood lead data and the need to evaluate the contribution of paint. In addition, during these meetings, a "decision tree" approach was

suggested that proposed different threshold levels (primary and secondary) for screening decisions, action decisions and land use patterns.

Findings from the three cities (Baltimore, Boston, and Cincinnati) of the Urban Soil Lead Abatement Demonstration Project (peer review scheduled for completion in late 1994) indicate that dust and paint are major contributors to elevated blood lead levels in children. Furthermore, preliminary findings suggest that any strategy to reduce overall lead risk at a site needs to consider not only soil, but these other sources and their potential exposure pathways. (For further information on this demonstration project, contact Dr. Rob Elias, USEPA/ORD, Environmental Criteria and Assessment Office (ECAO), RTP, (919) 541-4167.)

Finally, in its efforts to develop this interim directive, the OSWER Soil Lead Workgroup has met with other EPA workgroups including the TSCA Section 403, Large Area Lead Sites, and Urban Lead workgroups, as well as other Federal agencies including the Agency for Toxic Substances and Disease Registry, the Centers fo Disease Control, and the Department of Housing and Urban Development.

Derivation of Lead Screening Levels. Development of the residential screening level in this interim directive required two important OSWER decisions. 1) OSWER determined that it would seek to achieve a specific level of protectiveness in site cleanups; generally, OSWER will attempt to limit exposure to soil lead levels such that a typical (or hypothetical) child or group of similarly exposed children would have an estimated risk of no more than 5% of exceeding the a 10 µg lead/dl blood lead level. This 10 µg/dl blood lead level is based upon analyses conducted by the Centers for Disease Control and EPA that associate blood lead levels of 10 µg/dl and higher with health effects in children; however, this blood lead level is below a level that would trigger medical intervention. 2) In developing the residential screening level, OSWER has decided to apply the EPA's IEUBK model on a site-specific basis. This model has been designed specifically to evaluate exposures for children in a residential setting. Current research indicates that young children are particularly sensitive to the effects of lead and require specific attention in the development of a soil screening level for lead. A screening level that is protective for young children is expected to be protective for older population subgroups.

In general, the model generates a probability distribution of blood lead levels for a typical child or group of children, exposed to a particular soil lead concentration and concurrent lead exposures from other sources. The spread of the distribution reflects the observed variability of blood lead

levels in several communities. This variability arises from several sources including behavioral and cultural factors.

The identification of lead exposures from other sources (due to air, water, diet, paint, etc.) is an essential part of characterizing the appropriate blood lead distribution for a specific neighborhood or site. For the purpose of deriving a residential screening level, the background lead exposure inputs to the IEUBK model were determined using national averages, where suitable, or typical values. Thus, the estimated screening level of 400 ppm is associated with an expected "typical" response to these exposures, and should not be taken to indicate that a certain level of risk (e.g., exactly 5% of children exceeding 10 µg/dl blood) will be observed in a specific community, e.g., in a blood lead survey.

Because a child's exposure to lead involves a complex array of variables, because there is population sampling variability, and because there is variability in environmental lead measurements and background levels of lead in food and drinking water, results from the model may differ from results of blood lead screening of children in a community. Extensive field validation is in progress. The model will be evaluated further once these efforts are completed.

OBJECTIVE

With this interim directive, OSWER recommends using 400 ppm soil lead (based on application of the IEUBK model) as a screening level for lead in soil for residential scenarios at CERCLA sites and at RCRA Corrective Action sites. Residential areas with soil lead below 400 ppm generally require no further action. However, in some special situations, further study is warranted below the screening level. For example, agricultural areas, wetlands, areas with ecological risk, and areas of higher than expected human exposure are all situations that could require further study. For further guidance on ecological risks, Superfund risk managers are encouraged to consult their Regional Biological Technical Assistance Groups (BTAGs; see Appendix D).

Generally, the ground water pathway will not pose a significant risk since many lead compounds are generally not highly mobile. However, there are situations where, because of the form of lead, hydrogeology, or the presence of other contaminants at the site, lead may pose a threat to the ground water. In these situations, additional analysis is warranted, and the Superfund Regional Toxics Integration Coordinators (RTICs; see Appendix B) or RCRA hydrogeologists should be consulted.

While recognizing that urban lead is a significant problem, this interim directive is not designed to be applied in addressing the potential threat of lead in urban areas other than at CERCLA or RCRA Corrective Action sites. Guidance and regulations to be developed under TSCA Section 403 will provide an appropriate tool for addressing urban sites of potential concern.

Generally, where the screening level is exceeded, OSWER recommends using the IEUBK model during the Remedial Investigation or the RCRA Facility Investigation for evaluating potential risks to humans from environmental exposures to lead under residential scenarios. Site-specific data need to be collected to determine PRGs or MCSs. At a minimum, this may involve collecting soil and dust samples in appropriate areas of the site. Further guidance on data collection or modification of the non-residential equation can be obtained by contacting the RTICs or RCRA Regional risk assessors, who in turn may consult the Lead Technical Review Workgroup.

The type of site-specific data that should be collected will obviously depend on a number of factors, including the proximity of residences to the contaminated soil, the presence of site access controls, and other factors that would influence the probability of actual human exposure to the soils. At a minimum, when residences are at or near the site, it is expected that using the model will generally involve taking soil and dust samples from appropriate areas of the site. In many cases, it may not be necessary to gather certain types of data for input into the model. For example, when there are no residences nearby, or where there is otherwise no exposure or very limited exposure to lead contamination, it may not be necessary to collect site-specific data (e.g., dust, water, paint, blood-lead, etc.)

In developing a PRG for CERCLA sites or a MCS for RCRA facilities, EPA recommends that a soil lead concentration be determined so that a typical child or group of children exposed to lead at this level would have an estimated risk of no more than 5% of exceeding a blood lead of 10 µg/dl. In applying the IEUBK model for this purpose, appropriate site specific data on model input parameters, including background exposures to lead, would be identified.

When the PRG or MCS is exceeded, remedial action is generally recommended. Such action does not, however, necessarily involve excavating soil. A range of possible actions may be considered, as discussed in greater detail under the Implementation section of this directive: Issues for Both Programs.

or portions of sites do not require further study and to encourage voluntary cleanup. Screening levels are defined as a level of contamination above which there may be enough concern to warrant site-specific study of risks. Levels of contamination above the screening level would NOT automatically require a removal action, nor designate a site as "contaminated."

The residential screening level for lead described in this directive has been calculated with the Agency's new Integrated Exposure Uptake Biokinetic Model (IEUBK) model (Pub. # 9285.7-15-2, PB93-963511), using default parameters. As outlined in the Guidance Manual for the IEUBK Model for Lead in Children (Pub. # 9285.7-15-1, PB93-963510, February 1994), this model was developed to: recognize the multimedia nature of lead exposures; incorporate important absorption and pharmacokinetic information; and allow the risk manager to consider the potential distributions of exposure and risk likely to occur at a site (the model goes beyond providing a single point estimate output). For these reasons, this approach is judged to be superior to the more common method for assessing risks of non-cancer health effects which utilizes the reference dose (RfD) methodology. Both the Guidance Manual and the model are available to Superfund staff through the Superfund Document Center (703-603-8917) and to the public through the National Technical Information Service (703-487-4650).

Residential preliminary remediation goals (PRGs) for CERCLA remediations and media cleanup standards (MCSs) for RCRA corrective actions can be developed using the IEUBK model on a site-specific basis, where site data support modification of model default parameters. At some Superfund sites, using the IEUBK model with site-specific soil and dust characteristics, PRGs of more than twice the screening level have been identified. However, it is important to note that the model alone does not determine the cleanup levels required at a site. After considering other factors such as costs of remedial options, reliability of institutional controls, technical feasibility, and/or community acceptance, still higher cleanup levels may be selected.

The implementation of this guidance is expected to provide for more consistent decisions across the country and improve the use of site-specific information for RCRA and CERCLA sites contaminated with lead. The implementation of this guidance will aid in determining when evaluation with the IEUBK model is appropriate and in assessing the likelihood that environmental lead poses a threat to the public. Use of the IEUBK model in the context of this guidance will allow risk managers to assess the

contribution of different environmental sources of lead to overall blood lead levels (e.g., consideration of the importance of soil lead levels relative to lead from drinking water, paint and household dust). It offers a flexible approach to considering risk reduction options (referred to as the "bubble" concept) that allows for remediation of lead sources that contribute significantly to elevated blood lead. This guidance encourages the risk manager to select, on a site-specific basis, the most appropriate combination of remedial measures needed to address site-specific lead exposure threats. These remedial measures may range widely from intervention to abatement. However, RCRA and CERCLA have very limited authority to address interior exposures from interior paint. For a detailed discussion of the decision logic for addressing lead-contaminated sites, see the Implementation section and Appendix A.

Relationship to lead paint guidance. In addition, this interim directive clarifies the relationship between guidance on Superfund and RCRA Corrective Action cleanups, and EPA's guidance on lead-based paint hazards (discussed further in Appendix C). The paint hazard guidance will be issued to provide information until the Agency issues regulations identifying lead-based paint hazards as directed by Section 403 of the Toxic Substances Control Act (TSCA)[2]. Lead-based paint hazards are those lead levels and conditions of paint, and residential soil and dust that would result in adverse health effects.

The two guidance documents have different purposes and are intended to serve very different audiences. As a result the approaches taken differ to some degree. The lead-based paint hazard guidance is intended for use by any person who may be involved in addressing residential lead exposures (from paint, dust or soil.) It thus relates to a potentially huge number of sites, and serves a very broad potential audience, including private property owners or residents in addition to federal or state regulators. Much residential lead abatement may take place outside any governmental program, and may not involve extensive site-specific study.

This OSWER guidance, on the other hand, deals with a much smaller number of sites, being addressed under close federal regulatory scrutiny, at which extensive site characterization will have been performed before cleanup decisions are made. Thus, the RCRA and CERCLA programs will often have the benefit of much site-specific exposure information. This guidance is intended for use by the relatively small number of agency officials who oversee and direct these cleanups.

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[2]Title IV of TSCA (including section 403) was added by the Residential Lead-Based Paint Hazard Reduction Act of 1992 (Title X of the Housing and Community Development Act of 1992).

Both the TSCA Section 403 and OSWER programs use a flexible, tiered approach. The OSWER guidance sets a residential screening level at 400 ppm. As noted above, this is not intended to be a "cleanup level" for CERCLA and RCRA facilities, but only to serve as an indicator that further study is appropriate. The Section 403 guidance indicates that physical exposure-reduction activities may be appropriate at 400 ppm, depending upon site-specific conditions such as use patterns, populations at risk and other factors. Although worded somewhat differently, the guidances are intended to be similar in effect. For neither guidance is 400 ppm to automatically be considered a "cleanup level"; instead, it indicates a need for considering further action, but not necessarily for taking action. Neither is meant to indicate that cleanup is necessarily appropriate at 400 ppm. The greater emphasis in this OSWER guidance on determining the scope of further study reflects the fact that both CERCLA and RCRA cleanups proceed in stages with detailed site characterization preceding response actions in every case.

Above the 400 ppm level, the Section 403 guidance identifies ranges over which various types of responses are appropriate, commensurate with the level of potential risk reduction, and cost incurred to achieve such risk reduction. For example, in the range of 400 to 5000 ppm, limited interim controls are recommended depending, as noted above, on conditions at the site, while above 5000 ppm, soil abatement is recommended. This OSWER guidance does not include comparable numbers above 400 ppm; instead, as discussed above, it recommends the site-specific use of the IEUBK model to set PRGs and MCSs, when necessary. The remedy selection process specified in the National Contingency Plan (NCP) should then be used to decide what type of action is appropriate to achieve those goals.

In general, because the Section 403 guidance was developed for a different purpose and audience, OSWER does not recommend that it be used as a reference in setting PRGs and MCSs or in determining whether action at a particular site is warranted. (To put it another way, it generally should not be treated as a "to be considered" document or "TBC" under CERCLA.) The section 403 guidance is meant to provide generic levels that can be used at thousands of widely varying sites across the nation. The detailed study that goes on at CERCLA or RCRA sites will allow levels to be developed that are more narrowly tailored to the individual site. Nothing in the section 403 guidance discourages setting more site-specific levels for certain situations; in fact, it specifically identifies factors such as bioavailability that may significantly affect the evaluation of risk at some sites.

The IEUBK model. The Agency is further studying both the IEUBK model and analyses of epidemiologic studies in order to better develop the technical basis for rulemaking under TSCA

Section 403. The Agency intends to promulgate regulations under Section 403 setting health-based standards for lead in soil and dust. OSWER intends to issue a final soil lead directive once the TSCA Section 403 regulations are finalized. For additional information on TSCA Section 403 developments, call (202) 260-1866.

However, the Agency believes that risk managers (risk assessors, on-scene coordinators, remedial project managers, and other decision-makers at Superfund and RCRA sites) are currently in need of the best guidance available today. The Agency believes that the IEUBK model is the best available tool currently available for assessing blood lead levels in children. Furthermore, use of the IEUBK provides allows the risk manager to consider site-specific information that can be very important in evaluating remediation options. Therefore, using the latest developments in the IEUBK model and the collective experience of the Superfund, RCRA Corrective Action, and TSCA Section 403 programs, the Agency is offering this guidance and is recommending a residential screening level for Superfund and RCRA sites of 400 ppm.

BACKGROUND

Early OSWER guidance (1989-1991). Four guidance documents on soil lead cleanup were issued by OSWER during the period of 1989 to 1991:

1. September 1989, OSWER Directive #9355.4-02. This guidance recommended a soil lead cleanup level of 500 - 1000 ppm for protection of human health at residential CERCLA sites.

2. May 9, 1990. RCRA Corrective Action program guidance on soil lead cleanup. This guidance described three alternative methods for setting "cleanup levels" (not action levels) for lead in soil at RCRA facilities. One approach was to use levels derived from preliminary results of IEUBK model runs. The other two approaches were to use the range of 500 to 1000 provided in the 1989 directive on CERCLA sites, or to use "background" levels at the facility in question.

3. June 1990, OSWER Directive #9355.4-02A. Supplement to Interim Guidance on Establishing Soil Lead Cleanup Levels at Superfund Sites. This memorandum reiterated that the September 1989 directive was guidance and should not be interpreted as regulation.

4. August 29, 1991. This supplemental guidance discussed EPA's efforts to develop a new directive that would

accomplish two objectives: (1) account for the contribution from multiple media to total lead exposure; and, (2) provide a stronger scientific basis for determining a soil lead cleanup level at a specific site.

Development of the IEUBK Model for OSWER use. During the 1989-91 time period, use of the EPA IEUBK model was identified as the best available approach for accomplishing the objectives outlined in the August 1991 guidance. The model integrates exposure from lead in air, water, soil, dust, diet, and paint with pharmacokinetic modeling to predict blood lead levels in children (i.e., children 6 to 84 months old), a particularly sensitive population.

In the spring of 1991, OSWER organized the Lead Technical Review Workgroup to assist Regional risk assessors and site managers in both using the model and making data collection decisions at CERCLA and RCRA sites. The workgroup was composed of scientists and risk assessors from the Regions and Headquarters, including the Office of Research and Development (ORD), and the Office of Pollution Prevention and Toxic Substances (OPPTS).

In November 1991, the EPA Science Advisory Board (SAB) reviewed the scientific merits of using the IEUBK model for assessing total lead exposure and developing soil lead cleanup levels at CERCLA and RCRA sites. In general, the SAB found the model to be an important advance in assessing potential health risks from environmental contaminants. However, the SAB also recommended additional guidance on the proper use of the model.

In response to SAB concern over the potential for incorrect use of the model and selection of inappropriate input values both for default and site-specific applications, OSWER developed a comprehensive "Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children" (referred to in this interim directive as the "Guidance Manual"). This Guidance Manual assists the user in providing inputs to the model to estimate risks from exposures to lead. It discusses the use of model default values or alternative values, and the application of the model to characterize site risks. Use of the Guidance Manual should facilitate consistent use of the IEUBK model and allow the risk assessor to obtain valid and reliable predictions of lead exposure. The Lead Technical Review Workgroup has been collecting data to further validate the model and to update the Guidance Manual as needed.

Relationship to RCRA Corrective Action "Action" Levels. The approach for calculating a screening level for lead (including exposure assumptions), set forth in this Revised Interim Soil Lead Directive, supersedes the guidance provided for calculating

"action" levels set forth in Appendix D of the proposed Subpart S Corrective Action rule. In the July 27, 1990 RCRA proposal (55 *Federal Register* 30798), EPA introduced the concept of "action levels" as trigger levels for further study and subsequent remediation at RCRA facilities. In this respect, the current directive's "screening levels" are analogous to the proposed rule's "action levels." In the proposal, where data were available, action levels were developed for three pathways of human exposure to contaminants: soil ingestion, water ingestion and inhalation of contaminated air. Exposure assumptions used in the calculations were set out in Appendix D of the proposal. For the soil pathway, action levels were calculated two different ways depending on whether the contaminant in the soil was a carcinogen or a systemic toxicant. Although lead was listed in Appendix A of the preamble to the rule as a class B2 carcinogen, no action level had been calculated because neither a carcinogenic slope factor (SF) nor a reference dose (RfD) had been developed by the Agency. Although the guidance in Appendix D of the proposed Corrective Action rule remains in effect with respect to other hazardous constituents, this directive now allows for the development of the lead screening ("action") level using the IEUBK model.

Recent developments (1992-Present). Following discussions among senior Regional and OSWER management, the OSWER Soil Lead Directive Workgroup (composed of Headquarters, Regional and other Federal agency representatives) recommended in the spring of 1992 that a "two step" decision framework be developed for establishing cleanup levels at sites with lead-contaminated soils. This framework would identify a single level of lead in soils that could be used as either the PRG for CERCLA site cleanups or the action level for RCRA Corrective Action sites, but would also allow site managers to establish site-specific cleanup levels (where appropriate) based on site-specific circumstances. The IEUBK model would be an integral part of this framework. OSWER then developed a draft of this directive which it circulated for review on June 4, 1992. The draft set 500 ppm as a PRG and an action level for RCRA facilities in residential settings.

Following development of this draft, OSWER held a meeting on July 31, 1992 to solicit a broad range of views and expertise. A wide range of interests, including environmental groups, citizens and representatives from the lead industry attended. This meeting encouraged OSWER to think more broadly about how the directive would affect urban areas, how lead paint and dust contribute to overall risk, and how blood lead data could be used to assess risk. In subsequent meetings with the Agency for Toxic Substances and Disease Control (ATSDR) and the Centers for Disease Control (CDC), options were discussed on how to use blood lead data and the need to evaluate the contribution of paint. In addition, during these meetings, a "decision tree" approach was

suggested that proposed different threshold levels (primary and secondary) for screening decisions, action decisions and land use patterns.

Findings from the three cities (Baltimore, Boston, and Cincinnati) of the Urban Soil Lead Abatement Demonstration Project (peer review scheduled for completion in late 1994) indicate that dust and paint are major contributors to elevated blood lead levels in children. Furthermore, preliminary findings suggest that any strategy to reduce overall lead risk at a site needs to consider not only soil, but these other sources and their potential exposure pathways. (For further information on this demonstration project, contact Dr. Rob Elias, USEPA/ORD, Environmental Criteria and Assessment Office (ECAO), RTP, (919) 541-4167.)

Finally, in its efforts to develop this interim directive, the OSWER Soil Lead Workgroup has met with other EPA workgroups including the TSCA Section 403, Large Area Lead Sites, and Urban Lead workgroups, as well as other Federal agencies including the Agency for Toxic Substances and Disease Registry, the Centers for Disease Control, and the Department of Housing and Urban Development.

Derivation of Lead Screening Levels. Development of the residential screening level in this interim directive required two important OSWER decisions. 1) OSWER determined that it would seek to achieve a specific level of protectiveness in site cleanups; generally, OSWER will attempt to limit exposure to soil lead levels such that a typical (or hypothetical) child or group of similarly exposed children would have an estimated risk of no more than 5% of exceeding the a 10 µg lead/dl blood lead level. This 10 µg/dl blood lead level is based upon analyses conducted by the Centers for Disease Control and EPA that associate blood lead levels of 10 µg/dl and higher with health effects in children; however, this blood lead level is below a level that would trigger medical intervention. 2) In developing the residential screening level, OSWER has decided to apply the EPA's IEUBK model on a site-specific basis. This model has been designed specifically to evaluate exposures for children in a residential setting. Current research indicates that young children are particularly sensitive to the effects of lead and require specific attention in the development of a soil screening level for lead. A screening level that is protective for young children is expected to be protective for older population subgroups.

In general, the model generates a probability distribution of blood lead levels for a typical child, or group of children, exposed to a particular soil lead concentration and concurrent lead exposures from other sources. The spread of the distribution reflects the observed variability of blood lead

levels in several communities. This variability arises from several sources including behavioral and cultural factors.

The identification of lead exposures from other sources (due to air, water, diet, paint, etc.) is an essential part of characterizing the appropriate blood lead distribution for a specific neighborhood or site. For the purpose of deriving a residential screening level, the background lead exposure inputs to the IEUBK model were determined using national averages, where suitable, or typical values. Thus, the estimated screening level of 400 ppm is associated with an expected "typical" response to these exposures, and should not be taken to indicate that a certain level of risk (e.g., exactly 5% of children exceeding 10 µg/dl blood) will be observed in a specific community, e.g., in a blood lead survey.

Because a child's exposure to lead involves a complex array of variables, because there is population sampling variability, and because there is variability in environmental lead measurements and background levels of lead in food and drinking water, results from the model may differ from results of blood lead screening of children in a community. Extensive field validation is in progress. The model will be evaluated further once these efforts are completed.

OBJECTIVE

With this interim directive, OSWER recommends using 400 ppm soil lead (based on application of the IEUBK model) as a screening level for lead in soil for residential scenarios at CERCLA sites and at RCRA Corrective Action Sites. Residential areas with soil lead below 400 ppm generally require no further action. However, in some special situations, further study is warranted below the screening level. For example, agricultural areas, wetlands, areas with ecological risk, and areas of higher than expected human exposure are all situations that could require further study. For further guidance on ecological risks, Superfund risk managers are encouraged to consult their Regional Biological Technical Assistance Groups (BTAGs; see Appendix D).

Generally, the ground water pathway will not pose a significant risk since many lead compounds are generally not highly mobile. However, there are situations where, because of the form of lead, hydrogeology, or the presence of other contaminants at the site, lead may pose a threat to the ground water. In these situations, additional analysis is warranted, and the Superfund Regional Toxics Integration Coordinators (RTICs; see Appendix B) or RCRA hydrogeologists should be consulted.

While recognizing that urban lead is a significant problem, this interim directive is not designed to be applied in addressing the potential threat of lead in urban areas other than at CERCLA or RCRA Corrective Action sites. Guidance and regulations to be developed under TSCA Section 403 will provide an appropriate tool for addressing urban sites of potential concern.

Generally, where the screening level is exceeded, OSWER recommends using the IEUBK model during the Remedial Investigation or the RCRA Facility Investigation for evaluating potential risks to humans from environmental exposures to lead under residential scenarios. Site-specific data need to be collected to determine PRGs or MCSs. At a minimum, this may involve collecting soil and dust samples in appropriate areas of the site. Further guidance on data collection or modification of the non-residential equation can be obtained by contacting the RTICs or RCRA Regional risk assessors, who in turn may consult the Lead Technical Review Workgroup.

The type of site-specific data that should be collected will obviously depend on a number of factors, including the proximity of residences to the contaminated soil, the presence of site access controls, and other factors that would influence the probability of actual human exposure to the soils. At a minimum, when residences are at or near the site, it is expected that using the model will generally involve taking soil and dust samples from appropriate areas of the site. In many cases, it may not be necessary to gather certain types of data for input into the model. For example, when there are no residences nearby, or where there is otherwise no exposure or very limited exposure to lead contamination, it may not be necessary to collect site-specific data (e.g., dust, water, paint, blood-lead, etc.)

In developing a PRG for CERCLA sites or a MCS for RCRA facilities, EPA recommends that a soil lead concentration be determined so that a typical child or group of children exposed to lead at this level would have an estimated risk of no more than 5% of exceeding a blood lead of 10 µg/dl. In applying the IEUBK model for this purpose, appropriate site specific data on model input parameters, including background exposures to lead, would be identified.

When the PRG or MCS is exceeded, remedial action is generally recommended. Such action does not, however, necessarily involve excavating soil. A range of possible actions may be considered, as discussed in greater detail under the Implementation section of this directive: Issues for Both Programs.

IMPLEMENTATION

Superfund

This interim directive applies to all future CERCLA Remedial Investigation/Feasibility Study (RI/FS) work; this interim directive should generally not be applied at sites for which risk assessments have been completed. For removal sites, this interim directive recommends that decisions regarding removal actions be considered first by the Regional Decision Team (RDT). The RDT will then refer sites to the removal program for early action, as appropriate.

The approach in this interim directive helps meet the goals set by the Superfund Accelerated Cleanup Model (SACM) for streamlining remedial decision-making. (This streamlined approach is described in Appendix A, Suggested Decision Logic for CERCLA and RCRA Corrective Action.) This interim directive also recognizes that other methods (e.g., slope studies and others) for evaluating risks at lead sites may also be appropriate and may be used in lieu of, or in conjunction with, the IEUBK model. If an alternate approach to lead risk assessment is to be applied, an EPA scientific review should be obtained. For example, expert statisticians would need to review slope factor calculations for statistical biases before their use could be supported. Recognizing that all assessment methods involve some uncertainties, the Agency, at this time, believes the IEUBK model is the most appropriate and widely applicable tool for Superfund and RCRA sites. Alternatively, EPA may require setting cleanup levels below the screening level if site-specific circumstances warrant (e.g, ecological risk). For further information on the use of the IEUBK model at CERCLA sites, contact the Regional Toxics Integration Coordinators identified in Appendix B.

RCRA Corrective Action

It is expected that the RCRA corrective action program will generally follow an approach similar to CERCLA's (as described above) in using the IEUBK model. In the case of RCRA facilities at which lead contaminated soils are of concern, collection and evaluation of data for the purpose of using the model will be primarily the responsibility of the owner/operator.

Issues for Both Programs

Cleanup of soils vs. other lead sources: OSWER's approach to assessing and managing risks from lead is intended to address the multi-media/multi-source nature of environmental lead exposures because it is expected that people at or near CERCLA and RCRA Corrective Action sites will experience lead exposures from sources in addition to contaminated soil. In some instances, these other exposures may be large e.g., where there are

children living in houses with high levels of lead dust from deteriorated paint). The presence of various sources of lead exposure may be very important in both the development of site-specific risk assessments and in the consideration of alternative risk management options.

From an assessment perspective, estimating blood lead levels, that might result from exposures at a site, depends on appropriately integrating exposures from all relevant media. Specifically, it is important to consider direct soil exposures and indoor dust exposures (which can include contributions from both soil and lead-based paint) on a site-specific basis, as well as any contributions from drinking water or other local sources of lead exposure. In using the IEUBK model to estimate blood lead levels, it is important to note that the risk attributable to soil lead exposures is dependent upon the existing level of exposures from other sources. That is, the amount by which the total risk would be lowered if all exposures to lead in soil were removed is not a constant, but varies with the level of existing non-soil exposures. This is because the model derives a "distribution" (rather than a simple point estimate) as an output whose shape and size is quite dependent on the predicted variability of exposures from each lead source. As a result, other factors being equal, the risks attributable to soil will generally be higher in the presence of elevated lead exposures from other sources. Therefore, in applying the IEUBK model, the risk attributable to soil lead can be predicted as the difference between the risk estimated when all sources of lead exposure are assessed, and the risk estimated considering only non-soil related exposures. This concept is especially important when evaluating different options for risk reduction at a given site.

From a risk management perspective, achieving a safe environment for populations at CERCLA and RCRA Corrective Action sites may require attention to multiple sources of lead, not all of which may be related to contamination from the source that was the initial concern at the site. Generally, the goal of the Agency, while acting within the constraints of CERCLA and RCRA legal authorities, is to reduce, to the maximum extent feasible, the risk of having significantly elevated blood lead levels. On a site-specific basis this can include remediation approaches that would lead to reduction of exposure from other sources, such as lead-based paint, in conjunction with appropriate soil remediation. Following from the risk assessment discussion in the previous paragraphs, exposures from lead in soils may have a lesser impact in producing high blood lead levels if existing exposures from lead-based paint are reduced.

Abatement vs. Intervention: Remedial measures can be divided into those that remove the source of contamination (abatement) and those that leave the contamination in place but block the

exposure pathway (intervention). These combinations of measures might include but not be limited to:

Abatement - Soil removal or interior and exterior lead paint abatement.

Intervention - Institutional controls, education/public outreach, gardening restrictions, indoor cleaning and dust removal, or additional cover.

Generally, the most appropriate CERCLA or RCRA response action or combination of actions will be based, in part, on the estimated level of threat posed at a given site. However, as mentioned earlier, key decision criteria also include the overall protectiveness of response options, attainment of Applicable or Relevant and Appropriate Requirements (for CERCLA), a preference for permanent remedies, implementability, cost-effectiveness, and public acceptance. Intervention measures may be more appropriate than abatement (e.g., soil excavation) at many sites, especially in areas where soil lead levels fall at or near the site-specific PRG or MCS.

Addressing exposure from other sources of lead may reduce risk to a greater extent and yet be less expensive than directly remediating soil. In some cases, cleaning up the soil to low levels may, by itself, provide limited risk reduction because other significant lead sources are present (e.g., contaminated drinking water or lead-based paint in residential housing). If it is possible to address the other sources, the most cost-effective approach may be to remediate the other sources as well as, or (if exposures to lead in soil are relatively low) instead of full soil lead abatement.

Lead-based paint can be a significant source of lead exposure and needs to be considered when determining the most appropriate response action. Interior paint can contribute to elevated indoor dust lead levels. In addition, exterior paint can be a significant source of recontamination of soil. Appendix A-3 of this document contains more information on how to evaluate and address the contribution of paint.

Certain legal considerations arise in considering remediation of sources other than soil. In particular, interior exposures from interior paint generally are not within the jurisdiction of RCRA or CERCLA. In addition, where other sources are addressed, issues may arise regarding the recoverability of costs expended by the Agency, or the possibility of claims being asserted against the Fund where other parties are ordered to do the work.

As discussed above, in considering whether to address sources other than soil, it is necessary to consider the risk

that would remain from the lead in the soil. In some cases, after risks from other sources have been addressed, unrestricted exposure to soil could be allowed while still being protective (e.g., where the IEUBK model result was heavily affected by the other sources). In other cases, soil risks may still be high enough to require abatement, containment or institutional controls to prevent high levels of exposure. In such cases, before a conclusion is made that the overall remedy will be protective, institutional controls should be carefully studied to make sure that they will be implementable, effective in both the long-term and short-term, and likely to achieve community acceptance.

A potentially useful approach that can be considered in conjunction with other, more active measures in reducing blood lead levels is to develop and promote public education and awareness programs that focus on the causes and prevention of lead poisoning in children. EPA's Office of Pollution Prevention and Toxics (OPPT) provides information on abatement of lead-based paint by the homeowner as well as inexpensive preventive measures the public can take to reduce their exposure to lead. Additional research to evaluate the effectiveness of educational efforts in reducing lead exposures are needed to allow better evaluation of the usefulness of this option. Further, OPPT is assessing the effectiveness of various lead paint abatement options emphasizing low-cost methods. For additional information, contact the National Lead Information Center at 1-800-424-LEAD.

Mining-related sites: Both risk assessors and site managers should be aware that there are a number of factors that affect the relationship between soil lead concentrations and blood lead levels. These factors include the variability in soil lead contribution to house dust levels, or differences in the bioavailability of lead. See discussion in next section, Use of blood lead data, for assessing differences between measured and predicted blood lead levels.

Thus, for mining-related sites without significant past smelting/milling activity, this interim directive encourages further research for characterizing the potential impact of particle size and speciation on soil bioavailability.

Site managers and risk assessors are cautioned that most areas impacted by mining activities are also associated with present or historical smelting or milling operations. Generalizations regarding distinct differences between mining and smelting or milling sites should be avoided until adequate site history and characterization are complete.

Use of blood lead data: In conducting Remedial Investigations (RIs) for CERCLA or RCRA Facility Investigations (RFIs) for RCRA Corrective Action, the interim directive

recommends evaluating available blood lead data. In some cases it may be appropriate to collect new or additional blood lead samples. In general, data from well-conducted blood lead studies of children on or near a site can provide useful information to both the risk assessor and site manager. However, the design and conduct of such studies, as well as the interpretation of results, are often difficult because of confounding factors such as a small population sample size. Therefore, any available blood lead data should be carefully evaluated by EPA Regional risk assessors to determine their usefulness. The Guidance Manual discusses how to evaluate observed blood lead survey data and blood lead data predicted by the IEUBK model.

The Guidance Manual recommends that blood lead data not be used alone either to assess risk from lead exposure or to develop soil lead cleanup levels. During its review of the IEUBK model, the SAB supported this position by asserting that site residents may temporarily modify their behavior (e.g., wash their children's hands more frequently) whenever public attention is drawn to a site. In such cases, this behavior could mask the true magnitude of potential risk at a site and lead to only temporary reductions in the blood lead levels of children. Thus, blood lead levels below 10 µg/dl are not necessarily evidence that a potential for significant lead exposure does not exist, or that such potential could not occur in the future.

Non-residential (adult) screening level. EPA also believes there is a strong need to develop a non-residential (adult) screening level. The IEUBK model is, however, not appropriate for calculating this screening level since it is designed specifically for evaluating lead exposures in children. At this time, EPA is considering a few options for developing this screening level. Several adult models have recently become available. Developing a screening level by using any of them is likely to require significant additional work by the Agency. This work might include testing, validation, and selection of one of the existing models or development of its own model, both of which would require a considerable amount of time. Consequently this would probably be a long-term option. A short-term option would be to develop a screening level based on a simple approach that approximates the more complicated biokinetics in humans. This can serve in the interim while more sophisticated adult lead exposure assessment tools can be identified or developed.

NOTICE: Users of this directive should bear in mind that the recommendations in this document are intended solely as guidance, and that EPA risk managers may act at variance with any of these recommendations where site-specific conditions warrant, as has been noted above. These recommendations are not intended, and cannot be relied upon, to create any rights, substantive or procedural, enforceable by any party in litigation with the United States, and may change at any time without public notice.

Because this document and the related Guidance Manual are not legally binding either upon EPA or other parties, Agency personnel should keep in mind if they are questioned or challenged in comments on a proposed remedial plan, such comments must be considered and a substantive explanation must be provided for whatever approach is ultimately selected. For example, while the IEUBK model is recommended here, its use is not a regulatory requirement and comments on the model or its use should be fully considered.

APPENDICES

APPENDICES

Suggested Decision Logic for Residential Scenarios for CERCLA and RCRA Corrective Action

Step 1: Determine soil lead concentration at the site.

If soil lead is less than 400 ppm:
STOP, no further action is required, UNLESS special circumstances (such as the presence of wetlands, other areas of ecological risk, agricultural areas, shallow aquifers, or other areas of potentially high exposure) warrant further study.

If soil lead is greater than 400 ppm:
PROCEED to Step 2, UNLESS 400 ppm is selected as a cleanup goal based on consideration of all relevant risk management factors.

Step 2: Evaluate probable land use and develop exposure scenarios.

Step 3: Collect appropriate site-specific data based on selected scenarios.

For example, sampling data may include:

- Soil and dust (at a minimum), paint, water, and air
- For unique site situations, data on speciation and particle size, and behavioral activities may be required.

Available blood lead data:

- If blood lead data are available, consult the Guidance Manual and Regional Risk Assessor.
- If blood lead data are not available, Regional Risk Assessors and site managers should consider the appropriateness of conducting a blood lead study to supplement available data.

Step 4: Run the IEUBK model with site-specific data to estimate risk and evaluate key exposure pathways at the site.

- If blood lead data are available, compare the data to the model results

Step 5: Where risks are significant evaluate remedial options.

If lead-based exterior or interior paint is the only major contributor to exposure, no Superfund action or RCRA corrective action is warranted.

If soil is the only major contributor to elevated blood lead, a response to soil contamination is warranted, but paint abatement is not.

If both exterior lead-based paint and soil are major contributors to exposure, consider remediating both sources, using alternative options as described in Appendix A-2.

If indoor dust levels are greater than soil levels, consider evaluating the contribution of interior lead-based paint to the dust levels. If interior lead-based paint is a major contributor, consider remediating indoor paint to achieve a greater overall risk reduction at lower cost. (See Appendix A-2.)

NOTE: Available authority to remediate lead-based paint under CERCLA and RCRA is extremely limited.)

Step 6: If the IEUBK model predicts elevated blood leads, rerun the model using the site-specific parameters selected to reflect remedial options in Step 5 to determine site-specific PRGs or MCSs for soil.

Appendix A-2

Suggested Decision Logic for Lead-based Paint for CERCLA and RCRA Corrective Action

(If soil lead levels are below screening levels, lead-based paint could be addressed by authorities other than RCRA or CERCLA.)

If soil lead levels are above screening levels:

Step 1. Examine condition of exterior paint and determine its lead content, if any.

- If paint is deteriorated, assess contribution or potential contribution of paint to elevated soil lead levels through speciation studies, structural equation modelling, or other statistical methods.

Step 2. Evaluate potential for recontamination of soil by exterior paint.

Step 3. Remediate exterior paint only in conjunction with soil.

- Determine appropriate remediation based on risk management factors (e.g., applying the nine criteria), remediating the major contributor first.

Step 4. Examine condition of indoor paint and determine its lead content, if any.

- If indoor dust lead concentration is greater than outdoor soil lead concentration (because of contamination from both interior paint and outdoor soil), remediate indoor dust (e.g., through a removal action, or making HEPA-VACS available to community).

Step 5. Once the risk from indoor paint has been assessed, examine options to abate indoor paint (e.g., PRP, State, local, HUD) and consult TSCA Section 403 program for additional information and/or guidance.

Step 6. While RCRA and CERCLA have very limited authority regarding the cleanup of interior paint, the remedy may take into account the reduction of

total risk that may occur if interior paint is addressed by other means. Thus, for example, a Record of Decision (ROD) or Statement of Basis (SB) may recognize that interior lead-based paint is being addressed by other means, and narrow the response accordingly (possibly making this contingent on completion of the interior lead-based paint abatement effort.

Appendix B

Superfund Regional Toxics Integration Coordinators (RTICs)

	Telephone	FAX
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Peter Grevatt EPA Region 2 26 Federal Plaza New York, NY 10278	212/264-6323	212/264-6119
Reggie Harris EPA Region 3 (3HW15) 841 Chestnut Street Philadelphia, PA 19107	215/597-6626	215/597-3150
Dr. Elmer Akin EPA Region 4 345 Courtland St, NE EPA 9452 Atlanta, GA 30365	404/347-1586	404/347-0076
Erin Moran EPA Region 5 HSRLT-5J 77 West Jackson Street Chicago, Il 60604	312/353-1420	312/886-0753
Jon Rauscher EPA Region 6 6H-SR 1st Interst. Bank Tower 1445 Ross Ave. Dallas, TX 75202	214/655-8513	214/655-6460
David Crawford (Acting) EPA Region 7 Superfund 726 Minnesota Ave. Kansas City, KS 66101	913/551-7702	913/551-7063
Chris Weis EPA Region 8 8HWM-SR 999 18th St. Suite 500 Denver, CO 80202	303/294-7655	303/293-1230
Dan Stralka EPA Region 9 CWA 75 Hawthorne Street San Francisco, CA 94105	415/744-2310	415/744-1916
Carol Sweeney EPA Region 10 ES-098 1200 6th Avenue Seattle, WA 98101	206/553-4699	206/553-0119

Appendix C

Relationship Between the OSWER Soil Lead Directive and TSCA Section 403 Guidance

Since lead exposures occur through all media, a variety of Agency programs address lead under a number of statutes. Lead in soil is addressed under TSCA Section 403, the RCRA Corrective Action program, and CERCLA, each of which differs somewhat in the types of sites that apply and the types of standards that are used. These differences are primarily due to differences in the purposes of the programs and the authority granted by the statutes under which they are developed. Section 403 soil standards will apply only to residential soil and the current TSCA guidance is generic in nature, with the same standards applying on a nationwide basis. Given the wide applicability of Section 403, generic standards are used in the current guidance in order to reduce resource requirements, as compared to site-specific decisions which can involve expensive and time-consuming analyses. Required RCRA and CERCLA activities are determined on a site-specific basis. The agency's recommendations for evaluating RCRA Corrective Action and CERCLA sites are contained in the OSWER Interim Soil Lead Directive.

In all three of these programs, the Agency's approach is to consider soil lead in the context of other lead sources that may be present and contribute to the total risk. For example, TSCA Section 403 specifically requires the Agency to consider the hazards posed by lead-based paint and lead-contaminated interior dust, as well as lead-contaminated soil. Likewise, the OSWER Soil Directive includes evaluation of other lead sources at a site as part of site assessment/investigation procedures. In addition, the primary focus of the three programs is primary prevention -- the prevention of future exposures from the source(s) being remediated.

The fundamental difference between the relatively new TSCA Section 403 program and the RCRA Corrective Action and CERCLA cleanup programs is that, under current guidance the Section 403 program seeks to establish national standards to prioritize responses to lead hazards whereas the other two programs usually develop site-specific cleanup requirements. This is because TSCA Section 403 deals with a potentially huge number of sites, and resources for the investigation needed to accurately identify their risks are typically very limited. Therefore most decisions under Section 403 will be made with little or no regulatory oversight and clear generic guidelines will be more effective. The more established RCRA and CERCLA programs, on the other hand, deal with a much smaller number of sites, at which extensive site characterization will have been performed before cleanup decisions are made. In addition, these programs have well-established funding mechanisms.

Appendix D

Superfund Biological Technical Assistance Group Coordinators (BTAGs)

	Telephone	Fax
David Charters Mark Sprenger ERT USEPA (MS-101) 2890 Woodbridge Ave., Bldg. 18 Edison, NJ 08837-3679	908/906-6826	908/321-6724
Jeffrey Langhals TIB USEPA (52046) 401 M Street SW Washington, DC 20460	703/603-8783	703/603-9103
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Shari Stevens Surveillance Monitoring Branch USEPA Region 2 (MS-220) Woodbridge Avenue Raritan Depot Building 209 Edison, NJ 08837	908/906-6994	908/321-6616
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Shari Stevens Surveillance Monitoring Branch USEPA Region 2 (MS-220) Woodbridge Avenue Raritan Depot Building 209 Edison, NJ 08837	908/906-6994	908/321-6616
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MEMO TO: Tom Long

FROM: Maurice LeVois

DATE: July, 21, 1994

SUBJECT: RESPONSE TO COMMENTS OF U.S. EPA REVIEWERS REGARDING THE GRANITE CITY LEAD STUDY DRAFT REPORT

The subject review document is presented in two parts. The first part is a lengthy and detailed discussion, termed a "general issues" section. The second part of the review document contains specific comments. However, the 'general' comments section actually recapitulates all of the specific comments contained in the second half of the document. In order to facilitate understanding the relationship of the comments and responses, the responses presented below are numbered so as to correspond to the numbered comments in the general issues section.

1.1 Participation by zone has already been presented.

We made an extra effort to recruit in the high soil lead area - phone calls to residents started with this area, continued throughout the study.

1.2 The term "control group" implies that there is a clear definition of what, how, and why we are controlling by design or analysis strategy. In this case Pontoon Beach was different with respect to SES and living conditions (e.g. newer homes; a trailer park). They were not comparable to residents in our main study area (composed of old houses situated near the proposed cleanup area). Residents from neighboring areas of Granite City were far more comparable to our target group, and therefore, provided the best frame of reference for evaluating the effects of soil lead.

Use of a "control group" is actually an error in the design of studies of the effects of residential lead, unless it can be shown that the control group is like the study group in every respect except soil lead level. Our sample of subjects drawn from a more homogenous population spread over a distinct gradient of soil lead levels is the only sensible study design under these conditions.

1.3 Re-sampling of blood lead, combined with counselling intervention, resulted in a greater drop in blood lead than expected.

2.1 It was the EPA that developed the protocol for sampling house dust. The EPA developed criteria for evaluating the qualifications of prospective contractors, and selected the contractor who did the work. The EPA has all of the environmental data.

2.2 Again, the EPA developed the protocol for sample collection and QA/QC. The EPA selected and supervised the contractor that collected the samples. The EPA has the data.

2.3 Again, the EPA developed the protocol for sample collection and QA/QC. The EPA selected and supervised the contractor that collected the samples. The EPA has the data.

Unfortunately, some copies of our report were inadvertently distributed without an 'Appendix A' (the EPA soil collection protocol). That appendix is attached to this document as 'Attachment 1' to aid those reviewers who did not receive a copy of Appendix A earlier.

Ten soil samples were collected from the primary play areas in the yard around each house. No soil samples were taken from within the drip line of the house. A composite soil sample was made from the ten samples. This procedure should have yielded a representative soil sample from the yards and play areas. Since the great majority of the yards were very small, it is highly unlikely that the soil sampling protocol could have yielded unrepresentative soil lead results.

2.4 In both the inside (CI=1,2,3,4) and the outside (CO=1,2,3) rating of the condition of the house, the higher score was for the worst condition. This is a routine rating used by certified contractors specializing in lead paint inspection programs.

3.1 Although it is potentially useful to know that blood lead peaked in our study sample at around two years of age, the simple descriptive statistics that we present convey this information most directly. The simple graphic we present shows exactly at what age, and at what level, blood lead levels peak. It also shows the slope of the decline with age. Compared with this graphic presentation, the quadratic regression term recommended by the reviewer would have no meaning to most readers of the report.

Employing an nonlinear age covariate in blood lead regression models could increase slightly the amount of blood lead variance accounted for by age. That would have the effect of reducing slightly the amount of variance in blood lead remaining for other variables, such as soil and dust, to explain. However, including a quadratic expression for age would not appreciably change the overall blood lead R^2, nor would such a term improve our understanding of the influence of age or soil on blood lead.

3.2 The fact that our subjects lived in irregularly shaped residential areas, at varying distances form the closed smelter, is a strength, not a problem, in this study. None of our analyses, besides those involving distance from the smelter, depend in any way upon spatial location.

Soil lead is not uniformly distributed around the closed smelter either. Although soil lead levels decrease with distance from the closed smelter, there are hot spots and irregularities in the soil lead distribution throughout the study area. The sampling areas (zones 1--4) were used only to obtain a representative sample of homes and children across the entire range of soil lead levels, regardless of location. Neither distance, nor any other location variable, enters into the main multiple regression/correlation analysis - the point of which is to use the *joint distribution* of blood, soil, paint, dust, and water lead measures in the homes and yards of study participants, regardless of location, to understand how the variables are associated with one another.

The spatial distribution of blood lead is of interest because it can sometimes help to locate and explain clusters of high blood lead cases. That is why we depicted the physical location of the subjects in the study area. However, it was shown that distance is associated not only with soil lead and blood lead, but with SES, building condition, behavior, and other factors that influence blood lead. Simultaneous spatial depiction of all of these factors cannot be interpreted. That is the role of multiple regression/correlation analysis. The problem with the unadjusted bivariate tabulation presented by the reviewer in TABLE 1 of the EPA comments is that it totally ignores confounding by these other factors, which we have shown to be present.

3.3 This section presents a false and ridiculous argument. The reviewer took a meaningful linear multiple regression equation, mistakenly attempted to exponentiate the entire equation, and transformed it into a meaningless expression. The reviewer obviously misunderstood both the use of logs of the environmental and blood lead variables, and the meaning of the original regression equation.

First, it should be understood that the use of log-linear transformations made only a small difference in any of the analyses. However, since the environmental and blood lead measures were not normally distributed (they were skewed, with a few extreme high values), log transformation of the raw values resulted in more nearly normal distributions, and some improvement in the blood lead variance accounted for by the environmental measures. The methods used to analyze these continuous variables assume normal distributions of the variables, although the methods are robust enough to permit fairly radical departures from this assumption.

3.4 The statements in this section are also false, and indicate a lack of understanding of hierarchical regression. The reviewer incorrectly states that R^2 is not a "measure of effect", when the opposite is true.

> "..., one of the most attractive features of MRC is its automatic provision of proportion of variance and correlation measures of various kinds. These are measures of 'effect size,' of the magnitude of the phenomena being studied."
>
> Cohen and Cohen, in *Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences*, John Wiley & Sons, New York, 1975. p. 5-7.).

In our regression analysis of soil lead and blood lead we avoided including variables that could possibly confound the soil/blood lead relationship if including the other variables could over adjust (reduce) the size of the soil lead effect. The argument presented by the reviewer makes the incorrect assumption that including other variables might have increased the soil lead contribution. That is impossible. Every "adjustment" variable included in the regression model ahead of soil lead would necessarily account for some additional portion of the blood lead variance, thereby further reducing the variance left for soil to account for.

The reviewers do not appear to understand that the parameter estimates in our report (e.g in Table 10). Parameter estimates found at the final step in any stepwise multiple regression procedure capitalize on chance, and are not reliable. They should not be interpreted out of context. Stepwise procedures are only an aid in early exploration of the data, to be used along with careful consideration of the simple correlation matrix, and to be interpreted in the context of the earlier steps of the procedure, in which other variables enter and leave the equation.

The individual parameter estimates in any single step of a multiple regression model do not adequately express the adjusted contributions of the main study factors. In multiple regression, there is no substitute for set-wise hierarchical regression when attempting to adjust for possible confounding.

3.5 Pathway analysis as proposed is a subjective exercise that depends upon the assumptions of the analyst. We presented all of the descriptive statistics, bivariate statistics, and multivariate statistics used in our interpretation of the data. In particular, we described the importance of paint as a major contributor to dust lead in our study.

The point of Table 12 is missed entirely by the reviewer of this section, who misinterpreted the parameter estimates for paint,

dust, and soil presented in the second model. The correct interpretation of this analysis rests on the increment in R^2 when soil is added to Model 1.

In Model 1, water makes no difference, but it was one of our main environmental measures, and it cannot be viewed as possibly over adjusting the paint and soil effects, so we included it. Paint and building condition are obviously linked, as paint lead is much more likely to find its way into house dust, and to be available for ingestion, if the building is in poor condition. Paint and building condition account for 26% of dust lead variance. The addition of soil lead measures account for another 6% of dust lead variance, less than 1/4 the value of paint. Interpreting only the parameter estimates for the variables in Model 2 ignores the central meaning of the hierarchical analysis.

3.6 As stated above, adding behavioral or other variables to a hierarchical regression model can only reduce the variance accounted for by soil. The reviewer seems to want to find some set of variables that lead to a higher simultaneous parameter estimate for soil, regardless of how little variance is explained by the individual variables, or how all of the other environmental variables are affected by the factors the reviewer wants included in a single analysis. Such an analysis is meaningless. Behavioral variables can over adjust the effects of the main environmental variables, including soil, because behaviors are the pathways for environmental lead to reach the blood. It is incorrect to think that a better understanding of those variables can result from such an approach.

We have presented and discussed numerous bivariate relationships involving environmental, behavioral, and other factors, in order to show the considerable intercorrelation of these variables. Part of the point of that discussion was that we did not feel that it was possible to interpret multivariate analyses if we included all of these variables at once.

3.7 The comments in this section are correct, but this is exactly the opposite of the point of the preceding reviewer's comment (3.6)!

Our analysis avoided problems of multicollinearity by not including variables that could be proxies for one another. None of the variables included in the hierarchical regression models we presented are linked in this way.

3.8 While it is true that measurement error tends to reduce the magnitude of associations, this is equally true for all of the variables in this, and any other study. This does not change the relationship of the variables as long as the errors in measurement are not systematic.

We do not believe that there were systematic errors of measurement in this study. As stated above, we used a small set of key predictor variables, and did not have any problem with multicolinearity, or over adjustment of the soil lead effect.

4.1 We used 500 ug/g soil lead, and 10 ug/dl blood lead to conduct some two-group analyses, in addition to conducting other categorical and continuous data analyses. There were two reasons for conducting categorical analyses on these continuous data: 1. The ATSDR requested that we present part of our analysis in this way; 2. These cut points relate to a priori cleanup and blood lead levels set by EPA and CDC, respectively.

4.2 We agree that figures and graphs are helpful. Many more figures and graphs could be presented. However, the document is already quite long, and there is a limit to the amount of information that can be presented in this form. We presented figures and graphs when ever we thought that doing so would clarify a point of discussion.

4.3 The enlarged maps created by the reviewers indicates that our map was not as useless as stated, although we agree that reproduction of the original left a lot to be desired.

4.4 Confidence intervals can be estimated from the data provided, if it is thought by the reader to be important. We find little reason to believe that this is the case, since both the overall, and specific estimates of blood lead variance accounted for by the study factors is quite small in any event, and that is what really matters.

5.1 The facts speak for themselves. Our language choices differ. The majority of our higher blood lead values were not highly elevated (10.1-15). These slightly elevated levels were largely in children from relatively poor, unemployed families, living in run-down houses. Our interpretation is consistent with recommendations made by CDC in their [illegible] (pp. 7-21).

5.2 The reviewer does not understand that age was intentionally not used in the regression analysis (age was not "entered as a monotone predictor", as the reviewer states). This is because age is a proxy for exposure - through mouthing behavior that enables the ingestion of dust, paint, and soil. Adjusting the contribution of the environmental lead sources for dependence on age would clearly result in over adjustment, thus reducing the blood lead variance accounted for by the environmental measures. Note that only children under six were used in the analyses. While there is a wide range in the behavior of children in this age group, the play and mouthing behaviors that produce lead exposure are present over the entire range. That is why the 6 mos. - 6 years age group was the focus of this analysis.

5.3 The correlation of distance and blood lead was reported. There were other important correlations with distance that were also reported (e.g. parent's education, income, age and condition of the houses). Note that actual soil lead measures are used in the main regression analysis, not a proxy such as distance or location. A much better indication of the association of blood lead and soil lead is obtained by direct analysis of these two factors than can be gained by gerrymandering neighborhood subunits of the sample and speculation about clusters.

The comments here, as elsewhere in the review document, mistakenly focus on univariate and bivariate interpretation of soil lead associations, when the report makes clear that the soil lead data are confounded.

As noted above, the sampling zones were not used in the analysis. They were used only to draw a sample of households that spanned the full range of soil lead levels in an otherwise fairly homogeneous community. We directed extra effort at recruiting households from the central sampling zone in order to be sure we had adequate representation of the most highly (soil) exposed part of the population.

5.4 The EPA has such maps already, and can use the soil data they collected for this study to do additional mapping of the distribution of soil lead in the study area if that is their interest.

EPA soil lead maps were used as a basis for study subject selection. Those maps helped us to obtain a representative range of residential soil lead levels. However, as noted above, we used the joint distribution of soil lead levels measured in the subject's yards, along with other study variables, in our analysis of the predictors of blood lead. Actual soil lead level, not "radial distance", is the basis for our analysis and interpretation of the association of soil lead and blood lead.

5.5 This review comment is clearly false. We were very specific in our analysis of the contribution of dust lead to blood lead in our report, as well as in our analysis of the contribution of paint and soil to dust lead. It would have been a mistake to include dust lead in the analysis of soil and paint lead (as recommended by the reviewer). Since dust lead is almost entirely dependent on the lead in paint and soil, multicolinearity in the regression of all three environmental variables against blood lead could only produce a meaningless regression model.

5.6 The fact that ratings of overall building condition, as well as ratings of the immediate condition of paint at the point of XRF measurement increased the predictive value of paint measures supports our statement about the importance of this factor.

5.7 Inter-individual differences in behavior were important on an individual level. Such factors as hobbies and work related exposures were generally experienced by only a single family, and had no statistical value in the analysis. Important behavior-mediated exposures of this type must be considered on an individual basis, unlike paint and soil levels, which can be evaluated on a statistical level.

5.8 The speculation by the reviewer may, or may not be correct. The argument presented by the reviewer supports our decision not to include education, income, or other similar SES and behavioral factors in the main hierarchical regression model. It is not clear whether including these factors would correct for confound or over adjust the effects of the environmental measures.

5.9 This statement by the reviewer that our analysis cannot establish the contribution to blood lead of the environmental measures in our study is nonsense. That is exactly what our hierarchical analysis demonstrates.

6.0 We appreciate the list of names of individuals the EPA feels are qualified in this area. Dr. Kimbrough has had numerous discussions with Dr. Weitzman, one of the experts mentioned, and was involved in the initial stages of the design of the Urban Soil Lead Demonstration Project in Boston. Dr. Aschengrau was also involved in that study.

THE GRANITE CITY LEAD EXPOSURE DATASET: IEUBK MODELING AND EVALUATION OF SOIL LEAD AS A RISK FACTOR

GARY L. GINSBERG, Ph.D.
GALE F. HOFFNAGLE, CCM
TRC ENVIRONMENTAL CORPORATION

1/6/95

1.0 EXECUTIVE SUMMARY

The Draft Madison County Lead Exposure Study, Granite City, Illinois, (Illinois Dept. of Public Health; Institute for Evaluating Health Risks, February, 1994) represents an evaluation of blood lead concentrations in children who live in the vicinity of the former secondary lead smelter in Granite City. The study investigated the relationships between environmental/socioeconomic/behavioral factors and blood lead.

The draft report points out that blood lead concentrations in young children were not substantially impacted, with most of the children (total of 490 6 month to 6 year old children surveyed) having blood lead concentrations well below the USEPA level of concern (10 $\mu g/dl$). Additionally, blood lead concentrations were dependent upon a variety of factors, with the dependence upon soil lead apparently less significant than the dependence upon factors governing exposure to lead in house paint (e.g., paint lead XRF concentrations, building condition). A follow-up critique of the draft report by USEPA challenges these conclusions, and utilizes the Integrated Exposure Uptake Biokinetic (IEUBK) Model to justify a soil cleanup goal for Granite City of 400 to 500 ppm (Marcus, 1994).

The objectives of the current analysis are presented below together with a brief summary of our major findings. The detailed analysis supporting these findings is provided in subsequent sections.

Objective #1: Review and comment upon the results of the Draft Madison County Lead Exposure Study.

The blood lead distribution shown for Granite City children is typical of that expected for urban areas. While statistical analyses of the environmental lead/blood lead relationship are confounded by a variety of covariant parameters, soil lead is unlikely to be a major explanation for elevation in blood lead.

Objective #2: Evaluation of the accuracy of IEUBK Model predictions for Granite City.

The default model used by EPA to derive a soil lead cleanup goal (Marcus, 1994) is not predictive for the cases in which soil lead exceeds 500 ppm. The slope between

blood lead and soil lead (μg/dl change in blood lead per 1000 ppm change in soil lead) is overpredicted by approximately 4 fold by the default model.

Objective #3: Calibration of the IEUBK Model to better predict childhood blood lead concentrations at Granite City.

The best performance of the model is attained by decreasing the soil/house dust lead uptake (absorption) coefficient under conditions of high environmental concentrations. This calibration yields model predictions of the blood lead/soil lead slope that provide a good fit to the Granite City dataset. Note that TRC did not examine the algorithms embodied in the model and expresses no opinion as to their applicability in general.

Objective #4: Utilize the calibrated model to evaluate soil cleanup scenarios in terms of the possible benefits to community blood lead.

The calibrated model demonstrates that soil lead remediation to even very low concentrations (e.g., 200 ppm) would have only a slight impact upon blood lead as indicated by the limited effect of soil lead on indoor dust lead. Many children who have elevated blood lead do not live in elevated soil lead areas. The blood lead/soil lead slope factor relating blood lead to soil lead is thus shallow. Rather than focusing upon soil lead mitigation, a combined approach involving parental education, mitigation of strong lead sources (such as lead paint in poor condition, and grossly elevated soil and dust lead concentrations) may be the most effective approach, if it is decided that an intervention program of any kind is needed in this community.

Objective 5: Provide comments on the EPA critique of the Madison County draft report (Marcus, 1994).

EPA's critique focused upon a spatial relationship between blood lead and soil lead which is confounded by a variety of covariates. In the first instance, EPA appears to misinterpret the use of spatial correlation in the study. It was not intended as a method for comparing areas. Rather, it was simply intended to assure that a representative sample was obtained across the whole of the community. In any case, while soil lead and blood lead both decrease with increasing distance from the former smelter, the association between soil lead and elevated blood lead is weak and not statistically significant. Other environmental (particularly paint lead) and behavioral/ socioeconomic factors are likely stronger influences in creating blood lead exceedances. Further, EPA's use of the default IEUBK Model, which ignores the real data gathered at the site in contravention of the instructions stated in the user manual, has numerous flaws and provides a misleading assessment of the potential benefits of soil lead remediation. We believe that the cleanup scenarios presented in this analysis provide a more realistic representation of the effects of soil lead remediation.

2.0 REVIEW OF THE DRAFT MADISON COUNTY LEAD EXPOSURE STUDY

The draft report represents an evaluation of the relationship between environmental lead and blood lead levels in the community immediately surrounding the former smelter. The investigators obtained data on lead in soil, house dust, indoor and outdoor paint, and drinking water, and related these data to blood lead levels in 490 0 to 6 year old children. Smaller numbers of participants were included in the 6 to 15 year old and greater than 15 year old age groups. Additional factors considered for possible impact on childhood blood lead included: parental education and income level (socioeconomic status or SES), household number of cigarettes smoked per day, proximity to the former smelter, and age of the residence and its condition with respect to intactness of painted surfaces. Since a suitably matched control group was not identified, the study adopted a cross-sectional design relying upon regression analysis to test hypotheses regarding environmental lead: blood lead relationships. The study region was divided into concentric rings spreading outward from the former smelter to ensure a reasonably even spatial distribution of subjects, a point misunderstood by EPA.

The report provides important data on the blood lead distribution in the vicinity of the former smelter. For 0 to 6 year old children, the geometric mean blood lead was 5.58 $\mu g/d\ell$, with 16% of children having blood lead levels greater than 10 $\mu g/d\ell$. Only 7% of blood leads were above 15 $\mu g/d\ell$ in this age group. Co-linearity was found between key environmental risk factors such that soil lead, house dust lead, indoor/outdoor paint lead, condition of residence, parental income and educational level, and proximity to the former smelter site were correlated to one another. Therefore, the soil lead/blood lead relationship was confounded by a large number of interrelated variables. When hierarchical regression was used to account for key interrelated parameters (i.e., water lead, paint lead, condition of paint), it was shown that soil lead accounted for only 3% of the blood lead variance. In relation to other risk factors, the contribution of soil lead was considered to be quite small. For example, comparison of blood lead results across the soil lead <500 ppm vs. >500 ppm groups found only 1.4 $\mu g/d\ell$ differential. In contrast, a marked blood lead differential was found across residences representing different levels of upkeep. Blood lead in 0 to 6 year old children ranged from 6 $\mu g/d\ell$ when the residence was in good condition, to 8.2 $\mu g/d\ell$ for a rating of fair condition, to 11.8 $\mu g/d\ell$ for poor condition. Such findings lead to the conclusion that in this community, factors other than lead in soil have a more important impact on blood lead, in spite of the fact that soil lead levels ranged up to 3,000 ppm. Consistent with this is the results of an educational intervention in this community in which a marked blood lead decline was attributed to this intervention by the study authors.

Additional support for the concept that soil lead is not a key determinant of blood lead comes from the analyses provided below (Section 5, Figure 3) which show that the majority of blood lead exceedances in this community are in cases where soil lead is low (less than 500 ppm).

Since the Madison County Lead Exposure Study found that 16% of the 490 blood lead concentrations were above 10 $\mu g/d\ell$, there is a suggestion of a slight increase in community lead risk. The current USEPA criteria states that no more than 5% of children should be above 10 $\mu g/d\ell$.

While these blood lead data are a potential concern, it should be noted that the population geometric mean blood lead is not elevated (5.58 $\mu g/dl$ for 0 to 6 year old children), signifying that most children have normal blood lead. Additionally, the Granite City results are not materially different from what is found at urban areas where there is no former or current lead smelter. For example, the NHANES III dataset as compiled by Brody, et al. (1994), indicates that 16.4% of childhood (1 to 5 years old) blood lead values exceed 10 $\mu g/dl$ in urban areas of less than one million in population. This correspondence with Granite City blood lead results is striking and suggests that if a problem does exist at Granite City, it is best attributed to the same types of lead source that are typical of the urban environment (e.g., old housing containing dilapidated lead paint; historic lead fallout from fuel combustion). It should be noted that in the Granite City dataset, race had very little impact on blood lead, with the mean for white and non-white children not being statistically different. This contrasts with the NHANES III dataset where urban non-whites had substantially higher blood lead than did urban whites. It is possible that this indicates similar SES status for Granite City whites and non-whites since at Granite City, SES was a key determinant of blood lead.

3.0 ACCURACY OF THE DEFAULT IEUBK MODEL FOR PREDICTING GRANITE CITY BLOOD LEAD CONCENTRATIONS

Version 0.99d of the IEUBK Model was used with default parameter values (except for soil, dust and water lead where actual values were used) to provide predictions of childhood (0 thru 6 years old) blood lead levels. The model was run in the batch mode such that each record in the dataset could be put through the model and contribute individually to the overall statistics. Table 1 compares predicted and actual blood leads for the entire dataset encompassing 490 young children, and for subdivisions of the dataset based upon soil lead cutpoints. The model provided a reasonable fit to actual blood lead data for the entire dataset, both in terms of geometric mean blood lead and % greater than 10 $\mu g/dl$ (see top line: Total Population). However, the model overpredicted blood lead concentrations by nearly 2 fold in the soil lead subgroup that was greater than 1000 ppm (7.1 actual; 13.7 predicted). Additionally, the percentage of children with blood lead above 10 $\mu g/dl$ was overestimated by a large factor in this subgroup. A similar situation occurred in the 501 to 1000 soil lead subgroup, although the model overprediction was not as large (37%). In the lower soil lead groupings (0 to 250 ppm, and 251 to 500 ppm), the model-predicted blood lead was reasonably close to that actually observed, although in the lowest subgrouping, the model underpredicted by 27%.

Table 1 points out a major problem with default runs of the IEUBK Model for Granite City. The model predicts a soil lead/blood lead slope of 7.48, which is far above that actually seen (1.70). This overprediction of the slope leads to the false conclusion that blood lead is very sensitive to changes in soil lead such that if soil lead were remediated, blood lead levels should fall dramatically. The Urban Soil Lead Abatement Project (Baltimore, Cincinnati, Boston) indicated that very little benefit could be found after remediation of soil lead (e.g., Weitzman, 1993), which supports the concept of a low soil lead/blood lead slope. Other investigators have found similarly shallow soil lead/blood lead slopes (Starke, 1982; Yankel, 1977; Galke, 1975; Baltrop, 1975; Bornschein, 1990; Rabinowitz, 1988). Therefore, when modeling the benefit to be expected from soil lead remediation, it is critical

that the IEUBK Model be properly calibrated. Otherwise, the blood lead response to a change in soil lead will typically be overstated.

Note that TRC did not examine the algorithms embodied in the model and expresses no opinion as to their applicability in general.

4.0 CALIBRATION OF THE IEUBK MODEL FOR GRANITE CITY

The trend in Table 1 is that at low soil and dust lead concentrations (i.e., below 500 ppm), the model provides a good estimation of childhood blood lead. However, with increasing soil/dust lead concentration above 500 ppm, the model becomes increasingly overpredictive, such that for a significant percentage of young children at Granite City (29%), the default version of the model is inappropriate. The factor(s) which create serious model overprediction beginning at 500 ppm are not well defined, but it is clear that the model needs to be adjusted downward (i.e., less lead exposure and accumulation in blood) at the higher values for soil and dust lead. The most likely explanation for the overprediction may be decreased absorption of lead from soil and dust at higher lead loadings. This concept is consistent with a variety of literature sources (e.g., Sherlock, 1986; Bushnell, 1983) and is more plausible than other potential explanations (children contact less soil or house dust if it contains high lead; shift in lead internal distribution away from blood at higher intake).

On this basis, the model was fitted to the actual blood lead data by adjusting the model default soil/dust lead absorption coefficient (30% - total of saturable and non-saturable) to values that provide the best prediction of blood lead. The model was iterated using different absorption coefficients until a good fit was achieved for each soil lead subgrouping. Table 2 shows the back-fitted absorption coefficients that provide the best fit for several soil lead subgroupings. While the model default value of 30% soil absorption is appropriate for the 251-500 ppm group, lower absorption coefficients are required for fitting the model to actual data in higher soil lead groupings. The relationship between absorption coefficient and the composite soil/dust lead concentration approximates a straight line with a negative slope (lower absorption coefficient at higher soil leads) between 393 and 1213 ppm having a negative slope (Figure 1). This relationship can be used to approximate the lead absorption coefficient for any values of soil and dust lead in this community.

As shown in the next section, the calibrated model provided blood lead/soil lead slopes for cleanup scenarios that are reasonable estimates for that actually found at Granite City.

5.0 SOIL LEAD CLEANUP SCENARIOS

Table 3 utilizes the calibrated model to predict the blood lead benefit from remediating soil lead to 1000, 500, or 200 ppm in this community. All cleanup scenarios were run with actual batch file data for each household. Interior dust lead was adjusted to account for the remediation of soil lead by decreasing the dust lead by 0.7 times the decrement in soil lead. This approach is based upon USEPA's conservative assumption that 70% of the soil lead concentration is transferred to the indoor

environment to create house dust lead (USEPA, 1994). Our approach allows for house dust concentrations to exceed soil lead concentrations as is often the case at Granite City. The likely explanation for this differential is that interior lead sources (i.e., flaking interior paint) are a key source of dust lead. Thus, when soil lead is abated and nothing is done about interior lead paint sources, the house dust lead concentration will change by only that fraction contributed by soil lead. By adjusting dust lead by 0.7 times the decrease in soil lead, we are being faithful to the USEPA default for soil lead contribution to house dust while not ignoring other factors which contribute to house dust.

The data in Table 3 show the calibrated Model predictions of the benefit which could be expected from different soil lead cleanup targets (1000 ppm, 500 ppm, or 200 ppm). These data shown represent only the households which would be remediated, and do not take into account the overall impact on the community. This overall impact is presented in Table 4.

Table 3 indicates a modest decline in blood lead when soil leads are reduced to the indicated cleanup targets. The benefit of soil remediation is predicted to be greater above 1000 ppm (1.3 $\mu g/dl$ drop in geometric mean blood lead and 4 fold drop in percent of children with elevated blood lead) than below 1000 ppm, partially because the change in soil and house dust is larger at the higher starting concentrations. Additionally, as soil/house dust concentrations are lowered, the absorption coefficient is expected to increase thus decreasing the net effect on lead uptake. Thus, the table shows that diminishing returns are achieved by driving soil lead concentrations below 1000 ppm. It is important to note that even when soil lead is remediated to 200 ppm, the model predicts that the percentage of children with blood lead exceedances (i.e., >10 $\mu g/dl$) would still be high in the subgroup of houses that were remediated (13% above 10 $\mu g/dl$ in the 361 homes remediated to 200 ppm).

Table 4 indicates the blood lead benefit to be expected from the same three soil cleanup scenarios described in Table 3, but now data for remediated homes has been merged with the non-remediated homes (those below the remediation cutpoint) to determine the influence of remediation on the overall population (N=490 cases)blood lead. The influence of soil lead remediation on the overall population geometric mean is predicted to be miniscule, which is consistent with the fact that these cleanups would accomplish very minor reductions in population geometric mean soil and dust leads. Since the vast majority of households have soil and dust concentrations below 500 ppm, remediation of relatively few households at the top of the distribution shifts the overall exposure concentration little. Most importantly, soil remediation to concentrations as low as 200 ppm is modeled to produce only a small effect on the percentage of children with a blood lead in excess of 10 $\mu g/dl$.

It should be noted that these model simulations of cleanup scenarios yield a blood lead/soil lead slope ranging from 1.7 (Table 3) to 3.1 (Table 4). This is similar to the slope found in the current Granite City database (approximately 1.7) (Table 1). This demonstrates that the calibrated model is responsive to the actual blood lead/soil lead relationship and is likely to provide a good simulation of cleanup efficiency.

Figure 2 provides a frequency distribution of blood lead concentrations for the entire dataset of 490 cases. The observed line presents the data reported in the Madison County Lead Exposure Study. The predicted line for the 500 ppm (Pre500) cleanup scenario indicates a slight shift towards more children with blood lead values below 5 $\mu g/dl$, but with very little impact above 10 $\mu g/dl$. The 200 ppm remediation scenario (Pre200) indicates a larger shift, but with still a considerable number of children over 10 $\mu g/dl$.

The finding that soil lead remediation would have little beneficial effect on the community-wide rate of elevated blood lead reflects the fact that many children have elevated blood lead in spite of being surrounded by relatively low soil lead (below 500 ppm) (Figure 3). Remediation to 500 ppm will not affect these children, and it is unlikely that even lower soil remediation standards would be effective. For example, soil remediation will have little impact at homes whose house dust concentration clearly exceeds the soil lead concentration. Interior sources (e.g., paint lead) likely outweigh soil lead in such cases. The database contains 120 cases where house dust lead exceeds soil lead by 200 ppm or more, with 83 of these cases having at least a 500 ppm differential. Blood lead exceedances are a common occurrence in these cases (23%), and these cases will not be materially improved by soil lead remediation. Thus, based upon the blood lead exceedances distribution shown in Figure 3 and runs of the calibrated IEUBK Model (Tables 3 and 4), it is evident that soil lead remediation would be generally ineffectual in this community. A combined approach involving parental education, mitigation of strong lead sources (lead paint in poor condition; grossly elevated soil and dust lead concentrations) may be the most effective approach if it is decided that an intervention program is needed in this community.

6.0 EVALUATION OF EPA COMMENTS (MARCUS, 1994) ON THE MADISON COUNTY LEAD EXPOSURE STUDY

Mitigation of lead exposures is a worthwhile public health endeavor when properly directed towards high risk individuals or groups, and at the major causative factors contributing to blood lead (CDC, 1985). As pointed out above, the community as a whole does not appear to represent a high risk group for which lead mitigation would be especially required. The Madison County Lead Exposure Study and EPA's analysis suggest that subgroupings of this population may be at elevated risk in a manner which correlates with distance from the former smelter. The Madison County Lead Exposure Study points out the numerous confounding factors which affect the relationship between distance from the smelter and blood lead (e.g., year residence built, building condition, household income and education level, home ownership, soil lead). For each of these factors, residents were at greater risk (e.g., poorer building condition, lower family income and education, higher soil lead) as distance to the former smelter decreased. EPA attempts to show the relative importance of various lead sources to blood lead via correlational analyses involving spatial considerations (distance from smelter). Additionally, EPA provides a preliminary assessment of soil remediation options via the IEUBK model. However, EPA's assessment does not clearly differentiate between lead sources and provides no indication of their quantitative importance (e.g., soil lead/blood lead slope). Additionally, EPA's use of the IEUBK Model is flawed by arbitrarily assigning a default value for house dust when actual, site-specific house dust data are available. Further, the IEUBK

Model performs poorly for a large percentage of cases when model defaults (as used by EPA) are incorporated.

6.1 Assessment of Distance Rings

EPA suggests that soil lead is a more important contributor to blood lead than is paint lead based upon a distance ring subgrouping of the database. The Madison County Lead Exposure Study provided a parameter describing distance of each household from the former smelter. EPA correlated distance from the smelter with a variety of parameters which might impact blood lead to determine which factors seem most consistently associated with blood lead. Since paint lead concentrations don't vary with distance while soil lead, dust lead, and blood lead concentrations are correlated with distance, EPA suggests that soil lead rather than paint lead is the key contributor to dust and blood lead.

This correlational analysis, which focuses upon distance from the former smelter, is confounded by a variety of factors. The Madison County Lead Exposure Study demonstrates that such factors as year residence built, building condition, household income and education level, and home ownership confound the relationship between soil lead and blood lead as judged by distance. In fact, several of these factors would suggest that paint lead could become a stronger source of lead closer to the former smelter, in spite of the fact that paint lead levels are not actually correlated with distance. The fact that building condition worsens with proximity to the smelter suggests that paint lead would be more available to young children in homes nearer to the former smelter. Socioeconomic factors which affect children's exposure to lead paint sources (parental income, parental education, number of children per household) are all adversely affected with increasing proximity to the former smelter. Thus, the degree of parental supervision and awareness needed to prevent children's interaction with paint lead sources (e.g., gnawing on painted surfaces) appears to decline near the smelter. This concept is supported by the finding that children's mouthing of non-food objects increases in homes located near the smelter. These factors indicate that although paint lead levels are not correlated with distance, the degree of lead uptake from paint sources would still be expected to increase with increasing proximity to the former smelter. The likelihood that paint lead is substantially contributing to the blood lead vs. distance correlation is not recognized by EPA.

TRC examined the database subdivided by distance rings, to determine the strength of the association between soil lead and blood lead exceedances. If soil lead is an important causative factor in elevating children's blood lead above 10 μg/dl, one would expect that the frequency of blood lead exceedances at various distance rings would parallel the soil lead distribution across these rings. However, Figure 4 shows that this is not the case. This figure represents the data for distance rings 1 thru 9; distance ring 10 is not included because of the low number of cases (N=3), and distance rings 1 and 2 are combined because the number of cases in each group are relatively small (9 and 13, respectively), and because the soil and dust lead concentrations in these groups were nearly equal. It is clear from the figure that soil lead is not a major factor in elevating children's blood lead within these rings since only small changes in percent blood lead exceedances are seen (27% exceedance falls to 19%) over a soil lead range of 1000 ppm. In fact, regression of percent

blood lead exceedances in rings against the corresponding soil lead levels is not significant (p=.118; Figure 5).

In total, analysis of the dataset according to distance rings indicates a general tendency for soil lead and blood lead to decrease with increasing distance from the former smelter. However, the association between soil lead and the incidence of children with elevated blood lead is weak and not statistically significant. Other environmental (e.g., paint lead) and behavioral/socioeconomic factors are likely stronger influences in creating blood lead exceedances.

6.2 Critique of Soil Lead Remediation Goals Developed with the Default IEUBK Model

The EPA analysis concluded by back-calculating soil remediation goals based upon the default IEUBK Model. The major assumptions present in this exercise are analyzed below:

- House dust lead concentrations are 70% of soil lead concentrations, regardless of the actual house dust lead concentrations found in Granite City. EPA assumes that a soil lead-to-dust coefficient of 0.7 is appropriate because it obtained reasonable IEUBK batch mode predictions for Granite City children with this assumption. This validation exercise is flawed because it ignores the actual Granite City house dust lead data, and instead uses an incorrect default assumption. In fact, when the default model is run with the actual house dust lead concentrations, we find that in many cases (i.e., those over 500 ppm) it overpredicts blood lead concentrations (Table 1). By applying model defaults for house dust that are lower than the actual house dust data, EPA produces a reasonable fit, but one that has no basis in reality or scientific principles.

  The assumption that the Granite City environmental lead data can be described simplistically as house dust lead being 70% of soil lead is a significant error. (Note: The model's default contribution for airborne lead-to-house dust is insignificant). As the enclosed Table 1 shows, soil lead and house dust geometric means are overall, very similar. Thus, there is no basis to assume that dust lead is only 70% of soil lead. In fact, in numerous individual cases, dust lead levels far exceed the corresponding soil lead levels. Thus, while soil lead may influence house dust lead, other interior sources (e.g. lead paint) also play a fundamental role in driving dust lead. The model's remediation back-calculation option used by EPA does not allow the input of batch data files. Thus, household-specific data are lost in this exercise and an overly simplistic and incorrect model specification (house dust lead is 70% of soil lead with no significant interior sources) is introduced. This creates overestimates of the effectiveness of soil lead remediation because it ignores interior lead sources which are not affected by soil lead remediation. In other words, the assumption is that soil lead is fully responsible for house dust lead such that a 50% reduction in soil lead would yield essentially a 50% reduction in house dust lead. Obviously, if paint lead is the major contributor to house dust lead in particular homes, soil lead cleanup would have little impact on house dust or blood lead concentrations. Given the magnitude of the lead paint problem in Granite City and the number of cases in which house dust lead exceeds soil lead, the benefit of soil lead

remediation is substantially overpredicted by EPA's default back-calculation approach. This approach overlooks the well-known impact of paint lead on house dust lead. For example, Clark et al. (1985), have shown that housing stocks unlikely to contain lead paint can have low soil lead concentrations (average of 350 ppm found). However, in older homes, dust lead concentrations averaged 1,410 ppm and were two-fold higher if the home's condition was dilapidated. The USEPA Lead Criteria Document (1986) states that lead paint can be expected to substantially elevate dust lead from baseline levels.

We utilized a site-specific, non-default approach to estimate the benefit of soil lead remediation (Section 5.0). In this approach, the data from each household was run through IEUBK Model (batch mode) cleanup scenarios. It was conservatively assumed that 70% of the soil lead concentration is contributed to house dust lead, and that there are other interior sources that provide the remainder of the actual house dust lead measured. In our approach, soil lead remediation was modeled to remove that fraction of house dust lead that it is theoretically responsible for, while leaving in place that contributed by other sources (e.g., lead paint). This modeling approach is consistent with the IEUBK Model Guidance (USEPA, 1994; see pages 2-40 and 2-41), in which house dust lead is shown to consist not only of lead from soil, but also of lead from airborne deposition and from other unidentified interior (e.g., lead paint) and exterior (e.g., parental occupation) sources (the B_0 term in the house dust lead equation on page 2-41).

It should be noted that if one were to properly simulate house dust lead concentrations, the model offers a Multiple Source Analysis in which interior lead sources can be factored in. By using the default in this case (no interior sources), EPA misses an opportunity to render its use of the model more realistic. However, the best approach is still to run remediation scenarios in the batch file mode in which the actual house dust lead concentrations can be adjusted downward based upon the anticipated benefit from soil remediation. This is the approach we took in Section 5.0.

- Lead bioavailability from soil does not reflect the non-linearities observed at Granite City. The IEUBK Model assumes that the ½ saturation point for soil lead absorption is 100 μg lead ingestion/day. In other words, it would require a lead loading of 200 μg/day to begin to see a curvilinear (saturation) lead uptake response. With current model defaults, this level of lead loading would require a soil/house dust concentration of approximately 2,000 ppm. As discussed in Section 4.0, non-linearities which may involve saturable uptake occur at much lower concentrations. Therefore, the default version of the model is insensitive to the non-linearities seen at Granite City, and it must be modified if it is to be predictive of the benefits of soil lead remediation. The modifications described in Section 4.0 address these non-linearities, while the default approach used by EPA does not.

- A soil lead/blood lead slope of 7 to 8 μg/dl blood lead change per 1,000 ppm soil lead change. The model default creates this large slope by underestimating the non-linearities in the lead absorption profile, and by ignoring other factors which continue to add to blood lead

(e.g., paint lead, other lead sources) even when the soil lead concentration has been reduced. Thus, the benefit of soil lead remediation is grossly overpredicted by the default version of the model.

Our model runs, calibrated for the Granite City dataset using actual environmental data, provide a much more realistic soil lead/blood lead slope. The analysis provided by EPA is not site-specific and uses assumptions which overemphasize the influence of soil lead on blood lead. EPA's use of a default model that overpredicts the soil lead/blood lead slope by 4-fold (Table 1) indicates that the default model does not apply to Granite City. Our use of each household's data in validation runs and soil lead remediation scenarios provides a major advance over the default approach used by EPA.

7.0 REFERENCES

Baltrop, D.; Strehlow, C.D.; Thornton, I.; Webb, J.S. (1975) Absorption of lead from dust and soil. Postgrad. Med. J. 51: 801-804.

Bornschein, R.; Clark, S.; Pan, W.; Succop, P. (1990) Midvale community lead study, Final Report.

Bushnell, P.J.; DeLuca, H.F. (1983) The effects of lactose on the absorption and retention of dietary lead. J. Nutr. 113: 365-378.

Centers for Disease Control (CDC) (1985) Preventing Lead Poisoning in Young Children.

Clarke, C.S.; Bornschein, R.L.; Succop, P.; Que Hee, S.S.; Hamond, P.B.; Peace, B. (1985) Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. Environ. Res. 38: 46-53.

Galke, W.A.; Hammer, D.I.; Keil, J.E.; Lawrence, S.W. (1975) Environmental determinants of lead burdens in children. In: Hutchinson, T.C.; Epstein, S.; Page, Al. L.; Van Loon, J.; Davey, T.; eds. International conference on heavy metals in the environment: symposium proceedings, Vol. 3, October: Toronto, ON, Canada. Toronto, ON Canada: Institute of Environmental Studies, pp. 53-74.

Marcus, A.H. (1994) Preliminary Assessment of Data from the Madison County Lead Study and Implications for Remediation of Lead-Contaminated Soil.

Rabinowitz, M.B.; Bellinger, D.C. (1988) Soil lead-blood lead relationship among Boston children. Bull. Environ. Contam. Toxicol. 41: 791-797.

Sherlock, J.C.; Quinn, M.J. (1986) Relationship between blood lead concentrations and dietary lead intake in infants: the Glascow Duplicate Diet Society 1979-1980. Food additives and contaminants 3: 167-176.

Stark, A.D.; Quah, R.F.; Meigs, J.W.; DeLouise, E.R. (1982) The relationship of environmental lead to blood-lead levels in children. Environ. Res. 27: 372-383.

United States Environmental Protection Agency (EPA) (1986) Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children. EPA 540-R-93-081.

United States Environmental Protection Agency (EPA) (1986) Air Quality Criteria for Lead, Vol. III EPA 600/8-83/028aF-dF, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, NC.

Weitzmann, M.; Aschengrau, A.; Bellinger, D.; et al. (1993) Lead-contaminated soil abatement and urban children's blood lead levels. J. American Med. Assoc. 269: 1647-1654.

Yankel, A.J.; von Lindern, I.H.; Walter, S.D. (1977). The Silver Valley lead study: the relationship between childhood blood lead levels and environmental exposure. J. Air Pollut. Control Assoc. 27: 763-767.

NL INDUSTRIES/TARACORP SITE
COMMENTS TO PROPOSED PLAN

Prepared for:

NL Industries/Taracorp PRP Group

Prepared by:

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January 12, 1995

(B1229)

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NL INDUSTRIES/TARACORP SITE
COMMENTS TO PROPOSED PLAN
January 12, 1995

I. INTRODUCTION

In March 1990, the United States Environmental Protection Agency (USEPA) issued a Record of Decision (ROD) for the NL Industries/TaraCorp Superfund Site ("the Site") in Granite City, Illinois, in which the USEPA required a residential soil cleanup level for lead of 500 parts per million (ppm). Prior to issuing the final ROD, the USEPA accepted public comments. NL Industries, a PRP at the Site, provided such comments, which were incorporated into the Responsiveness Summary for the final ROD[1]. In October 1994, the USEPA issued a Proposed Plan that described the site background, site risks, and the residential soil cleanup level. The Proposed Plan noted that the USEPA is considering an amendment to the 1990 ROD and also invited public comments to be considered prior to choosing the final residential soil lead cleanup level for the Site.

As part of the process of determining a final residential soil lead cleanup level for the Site, the NL Industries/TaraCorp PRP Group ("the Group") is providing comments. The Group has retained McLaren/Hart Environmental Engineering Corporation (McLaren/Hart) to provide various technical comments on their behalf. The Group expects that these written comments will be considered as USEPA reviews and evaluates the residential soil cleanup level for lead and will be included in the Administrative Record.

On behalf of the Group, McLaren/Hart has conducted the activities described below.

- Reviewed the additional documents introduced into the Site's Administrative Record by the USEPA in support of the 500 ppm residential soil cleanup level for lead. (Attached as Exhibit A),

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[1] Since the time of the ROD, the USEPA has issued two Explanations of Significant Differences (ESDs) for the ROD (May 1993 and January 1994). Both ESDs were issued to allow the disposal of excavated materials off-site rather than consolidating them with the TaraCorp pile, substantially increasing remedial costs.

- Provided expert review (Attached as Exhibit B) and solicited expert review (provided separately as part of the Group's submittal of comments to the Proposed Plan) of the following documents:

  - "Madison County Lead Exposure Study, Granite City, Illinois," by R. Kimbrough, M. LeVois, and D. Webb (Illinois Department of Public Health);

  - "Comments on Madison County Lead Exposure Study, Granite City, Illinois," by A.H. Marcus, K. Hogan, P. White, and P. Van Leeuwen (USEPA);

  - "Response to Comments of the U.S.EPA Reviewers Regarding the Granite City Lead Study Draft Report," by M. LeVois (Illinois Department of Public Health); and

  - "Preliminary Assessment of Data from the Madison County Lead Study and Implications for Remediation of Lead-Contaminated Soil," by A.H. Marcus (USEPA),

- Reviewed the site-specific data and cleanup levels for other Superfund sites with lead contamination,

- Reviewed the transcript of the public meeting held on October 26, 1994 regarding the Site, and

- Reviewed the document entitled, "The Granite City Lead Exposure Dataset: IEUBK Modeling and Evaluation of Soil Lead as a Risk Factor" by G.L. Ginsberg, Ph.D. and G.F. Hoffnagle, CCM (TRC Environmental Corporation, January 1995).

Based on review of the above-referenced technical information regarding the development of soil lead cleanup levels and abatement of lead exposure, including documents relied upon by USEPA in the development of the Site residential soil cleanup level, McLaren/Hart has concluded that the USEPA cannot justify the selection of 500 ppm as the Site residential soil lead cleanup level and that, in fact, based on the data in hand, a cleanup level of 1,000 ppm is equally as protective of human health and the environment.

Specific comments related to the USEPA's selection of 500 ppm as the Site residential soil lead cleanup level are subsequently presented. The format for these comments is to provide a series

of major areas of concern with respect to the USEPA's development of the residential soil cleanup level. Within each area of concern, there is a discussion describing the basis for the concern.

II. COMMENTS ON THE PROPOSED PLAN

1. **In developing the residential soil lead cleanup level for the NL Industries/TaraCorp Site, the USEPA did not fully take into account the potential for sources of lead other than soil to impact blood lead levels. Further, the USEPA did not fully evaluate the potential that remediation of soils to the USEPA-selected residential soil lead cleanup level would not result in significantly decreased blood lead levels.**

- The presence of various sources of lead exposure was not adequately considered in the evaluation of alternative risk management options for the Site. However, as USEPA has noted in their document entitled "Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities" ("Interim Guidance," USEPA 1994b), estimating blood lead levels that might result from exposures at a site depends on appropriately integrating exposures from all relevant media.

- In the Interim Guidance, the USEPA notes that findings from the Urban Soil Lead Abatement Demonstration Project (USEPA 1993a, 1993b, 1993c, 1993d) indicate that any strategy to reduce the overall lead risk at a site needs to consider not only soil, but dust and paint as major contributors to elevated blood lead levels in children.

- The contribution of lead-based paint from the interior and exterior of homes has not been adequately considered in determining the most appropriate remedial response action for Granite City, although this approach is recommended by USEPA in their Interim Guidance (USEPA 1994b). In this guidance, the USEPA notes that the remedy for soil could be contingent upon completion of interior/exterior lead-based paint abatement effort.

- In the Interim Guidance, the USEPA notes that addressing exposure from other sources of lead may reduce risk to a greater extent and be more cost-effective than directly remediating soil. The USEPA further notes that, "In some cases, cleaning up the soil to low levels may, by itself, provide limited risk reduction because other significant lead sources are present (e.g., contaminated drinking water or lead-based paint in residential housing." Therefore, it is possible that applying a residential soil lead cleanup level of 500 ppm in the absence of addressing other lead sources will not achieve the desired reduction in blood lead levels, and remediating to 1,000 ppm would result in similar reductions in blood lead levels.

- The effectiveness of institutional controls and the development and promotion of public education and awareness programs that focus on the causes and prevention of lead poisoning in children needs to be more fully evaluated for Granite City. In the Interim Guidance, the USEPA notes that, after developing a preliminary remediation goal using the IEUBK model, and considering other factors such as costs of remedial options, reliability of institutional controls, technical feasibility, and/or community acceptance, still higher cleanup levels may be selected. In the Interim Guidance, the USEPA further

identifies that the development and promotion of public education and awareness programs focusing on the causes and prevention of lead poisoning in children, be considered in conjunction with active measures to reduce blood lead levels.

- Field studies have indicated that significant reduction (to less than 1,000 ppm) of lead in soil accessible to children would not result in a significant decrease in blood lead levels. Specifically, in the USEPA's Urban Soil Lead Abatement Demonstration Project in Baltimore, Maryland, the USEPA found that for every 1,000 ppm reduction of lead in soil, blood lead levels in children were reduced by only 1.0 microgram per deciliter (μg/dl). The results of this project lead USEPA to conclude that "Statistical analysis from the Baltimore Lead in Soil Project provides no evidence that the soil [lead] abatement has a direct impact on the blood lead level of children in the study." The USEPA further concluded that "in the presence of lead based paint in the children's homes, abatement of soil lead alone provides no direct impact on the blood lead levels of children." (USEPA 1993b).

2. **The USEPA's actions at the Site, including the selection of the residential soil lead cleanup level and the use of the IEUBK model in its selection, were inconsistent with USEPA's own most recent guidance for soil lead cleanup levels and for the application of the model.**

- It appears that site-specific data was not fully utilized by USEPA as an input into the IEUBK model to calculate a preliminary remediation goal (PRG) for the Site, although in its Interim Guidance, the USEPA states that in applying the IEUBK model for the development of a PRG, appropriate site-specific data on model input parameters, including background exposures to lead, be identified. If site-specific data was utilized, it is not clear as to the extent to which it has been utilized, and the site-specific data has not been included in the public record.

- USEPA's guidance manual for the use of the IEUBK model ("the IEUBK Guidance Manual," USEPA 1994c) states that if a blood lead study is to be evaluated in the risk assessment process, it is important that all of the sources of lead exposure at the Site be characterized and quantified. However, it appears that the USEPA only considered lead exposure via soil lead and resultant house dust. During the October 26, 1994 public meeting, Ms. Van Leeuwen, USEPA's representative, stated that lead paint was not considered in their modeling, only "environmental sources" (page 27 of the public meeting transcript, USEPA 1994d). However, as previously discussed, lead paint has been documented to be a significant source of blood lead, and the relative contribution of *all* sources of lead should have been evaluated.

- USEPA's Suggested Decision Logic for Residential Scenarios for CERCLA and RCRA Corrective Actions ("the decision logic," Appendix A-1 to the Interim Guidance) includes running the IEUBK model with site-specific data. If blood lead data are available, the evaluator is directed to compare that data to model results. A comparison of the model outputs to blood lead data was presented by the USEPA at the October 26, 1994 public meeting.

  Prior to running the model, the decision logic suggests that appropriate samples be collected, including soil, dust, paint, water, and air. While the condition of paint in residences was surveyed during the Pre-Design Field Investigation (conducted by Woodward-Clyde on behalf of the USEPA), no samples were collected. Any additional sampling and analyses which may have taken place has not been included in the public record.

  In the decision logic, USEPA further recommends that, where risks are significant, based on the output of running the IEUBK model, remedial options should be evaluated, including consideration of the relative risks of indoor and outdoor paint and dust.

  Finally, if the IEUBK model predicts elevated blood levels (under the existing conditions), the decision logic suggests that the model should then be re-run considering the implementation of abatement alternatives. In running the IEUBK model, however, USEPA did not consider any abatement measures other than the remediation of soils with lead above 500 ppm.

- The data presented by USEPA at the public meeting of October 26, 1994 included Figure 18 of the "Preliminary Assessment of Data from the Madison County Lead Study and Implications for Remediation of Lead-Contaminated Soil" by Allan Marcus (Marcus 1994b). Ms. Van Leeuwen discussed this figure and indicated during the public meeting that, "We think the model works pretty well. Especially at this site, it seems to have worked pretty well" (page 19 of the public meeting transcript, USEPA 1994d). When asked about the inclusion of lead paint contribution, she answered, "We only looked at the lead burden based on environmental exposures, not paint. Realizing that if paint was included, that those values would be higher" (page 27 of the public meeting transcript, USEPA 1994d) This evaluation of Figure 18 and explanation of not considering the contribution of paint are inconsistent. Because it is known that paint at many of the homes in Granite City contains high levels of lead. If paint were to be included, the good correlation between predicted and actual blood lead frequencies would not be demonstrated. In other words, including paint would significantly shift this predicted curve to the right, thus indicating the model is *not* valid for this Site when all exposures are accounted for. It was very misleading for the USEPA to claim this modeling approach worked well when all routes of exposure known to exist at the Site were not allowed to contribute to the predicted blood lead values.

- At the Bunker Hill Mining and Metallurgical Complex Superfund Site in Idaho (including the cities of Kellogg, Smelterville, Wardner, Pinehurst, and Page), also a smelter site (this was also a mining site), the USEPA selected a residential soil cleanup level of 1,000 ppm for lead. As indicated by Ms. Van Leeuwen during the public meeting, the effectiveness of soil removal to this cleanup level was demonstrated at the Kellogg Site (assumed to be the Bunker Hill Superfund Site, although not stated explicitly by Ms. Van Leeuwen), where children's blood lead levels dropped 8 μg/dl following soil removal. Given the effectiveness of soil remediation to 1,000 ppm lead at the Bunker Hill Site, it is possible that a residential soil lead cleanup level of 1,000 ppm would be equally as protective of human health and the environment at the NL Industries/TaraCorp Site.

3. **Even if the model is used for evaluation of a residential soil cleanup level, USEPA's application of the model for this purpose is flawed.**

- As discussed above, in both their Interim Guidance and in the IEUBK Guidance Manual, the USEPA recommends the use of site-specific data as inputs to the model. Although USEPA's representative stated during the public meeting that site-specific data for water, soil, and dust were utilized, the data utilized has not been included in the public record.

- During the public meeting of October 26, 1994, the USEPA representative was questioned by Mr. Tarpoff (p.25 of the public meeting transcript, USEPA 1994d) as to whether the model evaluated grass coverage or only bare soil as an input to the amount of soil ingested. The USEPA representative responded that the model used a set ingestion rate for each year of life from zero to seven. This approach is not consistent with the guidance set forth in the IEUBK Guidance Manual.

  USEPA's IEUBK Guidance Manual includes a section entitled "Are There Interrupted or Enhanced Exposure Pathways at the Site?" (Section 4.4.8.2). In that section, the USEPA cautions the model user against equating contaminant concentration with exposure or risk, where the risk assessor assumes *potential* exposure is *actual* exposure. The manual notes that if an exposure pathway is diminished or enhanced, the resulting exposure will also be diminished or enhanced. The manual provides the example that, at the same concentration of lead in soil, exposure to bare soil may be greater than if the soil has a good vegetation cover. Thus, in response to the Mr. Tarpoff's point above, the USEPA IEUBK Guidance Manual recognizes the variations in exposure (even specifically exposure to soil with varying degrees of vegetation) as a factor to be evaluated. However, during the public meeting, the USEPA representative implied that this factor was not taken into consideration in USEPA's modeling (page 25 of the public meeting transcript, USEPA 1994d). Had the USEPA considered the relative degree of vegetation cover, the blood lead levels predicted by the model would likely have been lower.

- As noted in Comment 2 above, the comparison of predicted and actual data in Figure 18 from the work of Allan Marcus (Marcus 1994b) indicates that the IEUBK model is *not* valid if all contributions to blood lead known at the site are considered.

- Ginsberg and Hoffnagle (1995) also demonstrated the shortcomings of the USEPA modeling approach. They ran the IEUBK default model and predicted geometric mean blood lead concentrations and percent of children with blood lead levels exceeding 10 μg/dl for the total Granite City population sampled and those with soil leads either above 1,000 ppm, 501 to 1,000 ppm, 251 to 500 ppm, or 0 to 250 ppm. The predictions were compared directly to the actual measured blood lead levels. The model provided reasonable results for the entire population, but overpredicted blood lead concentration and percent exceeding 10 μg/dl for the population with soil lead above 1,000 ppm and, to a lesser extent overpredicted the results for the 500 to 1,000 ppm group. The 251 to 500 ppm group was well predicted. The default model predicted a soil lead/blood lead slope of 7.48, which far exceeds the slope actually observed (1.70). These results indicate that the use of the default IEUBK model will lead to the false conclusion that blood lead levels are very sensitive to soil lead levels (which the actual data do *not* indicate). This further adds to the concern that the default model approach used by USEPA was flawed.

4. **If the IEUBK model is run utilizing site-specific parameters in evaluation of post-abatement conditions using ranges of soil lead cleanup levels, the results predict that addressing soil alone will not result in the USEPA's objective of less than 5 percent of children exceeding 10 μg/dl blood lead.**

- As stated in the Interim Guidance, the use of the IEUBK model is intended to allow USEPA to obtain valid and reliable predictions of lead exposure. However, data obtained as part of the design and implementation of the remedy selected by USEPA does not support the 500 ppm soil lead cleanup level. Utilizing site-specific data and a calibrated version of the IEUBK model, Ginsberg and Hoffnagle (1995) predicted that remediating soil even to very low levels (200 ppm) in the absence of other lead abatement efforts (e.g., for lead paint) will not have a significant impact on blood lead levels. They found very little benefit going from 1,000 ppm to 200 ppm when comparing blood lead predictions and the percent of children with blood lead levels exceeding 10 μg/dl.

5. **The technical documents placed in the Administrative Record do not support the 500 ppm cleanup level for lead in soils as a mechanism for mitigating potential health risks associated with the Site. However, several of the documents do support the need to evaluate and abate sources of lead in addition to soil to reduce blood lead levels.**

A review of the documents placed into the Administrative Record for the NL Industries/TaraCorp Site suggests that the majority of these references falls into one of three general categories: toxicological issues surrounding lead, statistical and modeling techniques for analysis of lead data, and issues surrounding lead-containing soils. McLaren/Hart has completed a review of these documents on behalf of the Group. The results of this review are detailed in a separate document entitled, "Review of Public Record Documents for the NL Industries/TaraCorp Site." McLaren/Hart's review is provided as Exhibit A to this document, and the results of the review are summarized briefly below.

- The technical documents reviewed that primarily addressed toxicological issues (i.e. health effects, biomarkers, metabolism/pharmokinetics of lead) indicated the following:

  - only a small percentage of this category of documents individually evaluated lead in soils as a source;

  - the effects of lead toxicity reported were primarily neurological, the source of lead was principally unspecified or of a multi-media source (paint, soil, dust air, diet);

  - when identifying the specific source of lead exposure in infants, children and adults, the documents reviewed cited leaded gasoline, lead-based paint and food as primary sources of lead intake and lead toxicity;

  - the bioavailability of lead in soil was reported to be lower than the bioavailability of lead from dietary lead sources due to particle size, solubility and lead speciation.

- The technical documents reviewed that primarily addressed statistical/modeling issues indicated the following:

  - using structural equation modeling, it was concluded that exterior deteriorating lead-based paint contributes significantly to the household dust fraction of lead via its presence in soils; and,

  - the complexity and variability associated with the analysis of lead data require consideration of a variety of factors ranging from scientific to socioeconomic issues in order to establish a cleanup level and potential remedial actions.

- The technical documents that primarily addressed issues related to lead in soils indicated the following:

  - lead in paint and dust have a significant impact on lead exposure;

- lead-based paint remains the most common high-dose source of lead exposure in preschool children;

- the Urban Soil Lead Abatement Demonstration Project authorized under SARA in 1986 was inappropriately used by USEPA during the October 26, 1994 public meeting to present their position that soil abatement plays a significant role in minimizing lead exposure; However, as discussed under Comment 1, the USEPA also concluded from the Urban Soil Lead Abatement Demonstration Project that abatement of one source of lead exposure should only be considered in the context of the other sources present.

- The majority of the technical documents reviewed supports the concept that PRGs (cleanup levels) should be determined for each site using site-specific data along with available data analysis techniques and current toxicological information. Consideration and integration of health-related socio-economic, cost, institutional control and public awareness and education factors must be weighed as part of the process of establishing a cleanup level for lead in soils.

As noted in the Section I of this document, McLaren/Hart solicited and provided expert review of the following documents that were placed in the Administrative Record:

- "Madison County Lead Exposure Study, Granite City, Illinois," by R. Kimbrough, M. LeVois, and D. Webb (Illinois Department of Public Health);

- "Comments on Madison County Lead Exposure Study, Granite City, Illinois," by A.H. Marcus, K. Hogan, P. White, and P. Van Leeuwen (USEPA);

- "Response to Comments of the U.S.EPA Reviewers Regarding the Granite City Lead Study Draft Report," by M. LeVois (Illinois Department of Public Health); and

- "Preliminary Assessment of Data from the Madison County Lead Study and Implications for Remediation of Lead-Contaminated Soil," by A.H. Marcus (USEPA).

Expert reports were prepared by McLaren/Hart (Exhibit B) and by Dr. Ellen J. O'Flaherty of the University of Cincinnati Medical Center (provided separately as part of the Group's submittal of comments to the Proposed Plan). In their assessment of the above-listed documents, these reports support the conclusion that the technical documents placed in the Administrative Record do not support the 500 ppm cleanup level for lead in soils as a mechanism for mitigating potential health risks associated with the Site. Specifically,

- The Madison County Lead Exposure Study, Granite City, Illinois (Illinois Department of Public Health 1994a), was of high quality and its conclusions were supported by the data and the statistical analyses performed.

- The USEPA predictions shown in Figure 18 of the "Preliminary Assessment of Data from the Madison County Lead Study and Implications for Remediation of Lead-Contaminated Soil," (Marcus 1994b) all exceed the observed values for the upper 20 percent of the data. This suggests that the lognormal model may be too heavy-tailed for this data. At the 95th percentile, blood lead levels were overpredicted by 4 μg/dl (25 percent), and at the maximum, the model overpredicted by about 70 μg/dl or more than 100 percent of the observed maximum. These evaluations indicate the IEUBK modeling and prediction performed by the USEPA for this Site did not fit the blood lead data as closely as the USEPA has indicated.

6. While USEPA was relying on current guidance at the time of the ROD in selection of the residential soil lead cleanup level, since that time, the USEPA has taken a more site-specific approach to the selection of cleanup levels at similar sites. In some cases, this has resulted in the selection of soil lead cleanup levels substantially higher than the 500 ppm level set for the NL Industries/TaraCorp Site.

- As indicated in Appendix B to the ROD, the selection of the soil lead cleanup level for the NL Industries/TaraCorp Site was based, in part on the USEPA's "Interim Guidance on Establishing Soil Lead Cleanup Levels" (USEPA 1989). This guidance relies on the Centers for Disease Control (CDC) recommendation that "...lead in soil and dust appears to be responsible for blood levels in children increasing above background levels when the concentration in the soil or dust exceeds 500 to 1,000 ppm." The USEPA further states that "Site-specific conditions may warrant the use of soil cleanup levels below the 500 ppm level or somewhat above the 1,000 ppm level."

  Since the time of the ROD, however, the USEPA has utilized site-specific information in the selection of soil lead cleanup levels higher than that selected for the NL Industries/TaraCorp Site. For example, at the Bunker Hill Mining and Metallurgical Complex Superfund Site (Bunker Hill) in Idaho (including the cities of Kellogg, Smelterville, Wardner, Pinehurst, and Page), also a smelter site (this was also a mining site), the USEPA selected a residential soil lead cleanup level of 1,000 ppm following such an approach. In the ROD for that site (USEPA 1991), the USEPA indicated that predicted lead intake was based on total lead levels from all media, and that the principal pathways were diet, drinking water, air, soils and dusts. While the ROD for the Bunker Hill site indicates that the assumptions on which the 1,000 ppm level were based may not apply to other sites, given the similarity of the sources of contamination between the Bunkder Hill site and the NL Industries/TaraCorp Site, it seems likely that a 1,000 ppm residential soil lead cleanup level would also be protective of human health and the environment at the NL Industries/TaraCorp Site.

- The USEPA has developed and published revised guidance for the development of lead cleanup levels (USEPA 1994b). This guidance supersedes the 1989 guidance, and allows for the consideration of site-specific conditions, including evaluation of exposure to environmental lead other than soils (e.g., paint, dust). As discussed under prior comments in this document, if the residential soil cleanup levels for the NL Industries/TaraCorp Site were developed following the current guidance, a higher residential soil lead cleanup level would likely be selected.

- In the ROD for the Bunker Hill site, the USEPA notes that, "The effectiveness of the 1,000 ppm threshold level for yard soils is dependent on several assumptions regarding reduced intakes along other pathways. Some of those assumptions are based on assessments of other remedial activities on the site and substantial reductions in dietary intake achieved from nationwide lead reduction initiatives." This approach, which includes evaluation of other remedial activities at the site, is consistent with the USEPA's more recent guidance, and should be followed for the selection of a residential soil lead cleanup level for the NL Industries/TaraCorp Site. Other remedial activities that should be taken into consideration for the NL Industries/TaraCorp Site should include the benefit from remedial activities in the industrial areas (e.g., covering the TaraCorp Pile), and the potential need for and exposure reduction from addressing other environmental sources of lead such as lead paint in the interior and exterior of homes.

III. SUMMARY AND CONCLUSIONS

As part of the process to determine a final residential soil lead cleanup level for the Site, technical comments on the Proposed Plan have been prepared for submittal to the USEPA and for inclusion in the Administrative Record. In the preparation of these technical comments, the Group conducted the following major activities:

- Completed a technical review of approximately 115 documents representing new information placed in the Administrative Record by USEPA;
- Solicited and provided expert review of four documents related to the Madison County Lead Exposure Study;
- Attended the public meeting of October 26, 1994 and reviewed the transcript from the meeting;
- Reviewed a document regarding USEPA's use of the IEUBK model on the lead exposure data for the Site (Ginsberg 1995); and
- Reviewed and evaluated USEPA guidance documents regarding the IEUBK model and the development of soil lead cleanup levels at Superfund and RCRA sites.

The major conclusions drawn from these activities are as follows:

- **In proposing a 500 ppm lead soil cleanup level, USEPA did not adequately consider the potential for sources of lead exposure other than soil to impact blood lead levels in children. Such an approach is flawed and does not appropriately consider the findings of technical documents newly placed in the Administrative Record, USEPA's most recent guidance on the development of soil lead cleanup levels, and USEPA's guidance on the use of the IEUBK model.**
- **The technical documents and USEPA's guidance regarding the development of a soil lead cleanup level do not support USEPA's conclusion that remediation of soils to 500 ppm lead would result in significantly decreased blood lead levels. USEPA's own Urban Soil Lead Abatement Demonstration Project provided no evidence that soil lead abatement has a significant direct impact on blood lead levels in children. Given the results of USEPA's work at the Bunker Hill Mining and Metallurgical Complex in Idaho, where USEPA stated that soil lead abatement was effective in reducing blood lead levels, a 1,000 ppm soil lead cleanup level would be protective of human health and the environment (1,000 ppm was the residential soil lead cleanup level selected for the site by the USEPA.)**

- **The USEPA did not follow its most recent guidance on developing soil lead cleanup levels and guidance on utilizing the IEUBK model. If USEPA's guidance were followed and the IEUBK model utilized in conformance with USEPA's guidance, a higher soil lead cleanup level (greater than 500 ppm) may be demonstrated to be equally protective of human health and the environment.**

- **The IEUBK model, as utilized by USEPA to justify a 500 ppm soil lead cleanup level, did not fully incorporate site-specific data or consider other important potential sources of lead exposure to children. These sources include lead paint, and site-specific measurements of lead levels in interior dust.**

- **The magnitude of the potential lead exposure problem predicted by USEPA's results from the IEUBK model (which did not fully utilize site-specific data) is inconsistent with the actual results of the blood lead study in children conducted by the Illinois Department of Public Health. While the USEPA has taken issue with some of the conclusions of the blood lead study, expert review has shown that its conclusions are sound, and can be relied upon in the evaluation of the outputs of the USEPA's modeling.**

Based on the review of new information provided by USEPA in its Administrative Record (including guidance developed by USEPA since the 1990 ROD regarding development of soil lead cleanup levels) and a more appropriate running of the IEUBK model; the Group strongly believes that USEPA should reconsider its residential soil lead cleanup level of 500 ppm. The Group's review of the information in the Administrative Record concludes that a residential soil cleanup level of 1,000 ppm could be utilized and still be protective of human health and the environment. Utilizing a residential soil lead cleanup level of 1,000 ppm would provide a better balance of trade-offs between remedial actions that could be implemented at the Site. A remedy that includes a 1,000 ppm residential soil cleanup level would be equally protective of human health and the environment, more implementable, and more cost-effective than the USEPA's selected remedy, including a 500 ppm residential soil cleanup level.

IV. REFERENCES

Ginsberg, G.L., Ph.D. and G.F. Hoffnagle, CCM (TRC Environmental Corporation). 1995. *The Granite City Lead Exposure Dataset: IEUBK Modeling and Evaluation of Soil Lead as a Risk Factor.* January 6.

Illinois Department of Public Health. 1994a. *Madison County Granite City, IL Lead Exposure Study.* February.

Illinois Department of Public Health. 1994b. *Response to Comments of the USEPA Reviewers Regarding the Granite City Lead Study Draft Report Study.*

Marcus, Allan H., Hogan, K., White, P., and Van Leeuwen, P. (USEPA). 1994a. *Comments on Madison County Lead Exposure Study, Granite City, IL.* May 23.

Marcus, Allan H. (USEPA). 1994b. *Preliminary Assessment of Data from the Madison County Lead Study and Implications for Remediation of Lead-Contaminated Soil.* Environmental Criteria and Assessment Office. October 14.

United States Environmental Protection Agency (USEPA). 1989. *Interim Guidance on Establishing Soil Lead Cleanup Levels at Superfund Sites.* OSWER Directive #9355.4-2. September 7.

United States Environmental Protection Agency (USEPA). 1990. *Record of Decision. NL Industries/TaraCorp, Granite City, Illinois.* March 29, 1990.

United States Environmental Protection Agency (USEPA). 1991. *Record of Decision. Bunker Hill Mining and Metallurgical Complex, Idaho.* August 30, 1991.

United States Environmental Protection Agency (USEPA). 1993a. *Review Draft: Urban Soil Lead Abatement Demonstration Project: Volume I: Integrated Report.* Office of Research and Development, EPA/600/AP-93-001A. July.

United States Environmental Protection Agency (USEPA). 1993b. *Urban Soil Lead Abatement Demonstration Project: Volume II: Boston Report. Office of Research and Development*, EPA/600/AP-93-001B. July.

United States Environmental Protection Agency (USEPA). 1993c. *Urban Soil Lead Abatement Demonstration Project: Volume III: Baltimore Report.* Office of Research and Development, EPA/600/AP-93-001C. July.

United States Environmental Protection Agency (USEPA). 1993d. *Urban Soil Lead Abatement Demonstration Project: Volume IV: Cincinnati.* Office of Research and Development, EPA/600/AP-93-001D. July.

United States Environmental Protection Agency (USEPA). 1994a. *Proposed Plan: NL Industries/TaraCorp Superfund Site, Granite City, Illinois.* October 13.

United States Environmental Protection Agency (USEPA). 1994b. *Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities.* OSWER Directive #9355.4-12. July 14.

United States Environmental Protection Agency (USEPA). 1994c. *Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children. EPA-540/R-93/081.* February 14.

United States Environmental Protection Agency (USEPA). 1994d. *Transcript of October 26, 1994 Public Meeting.*

THE GRANITE CITY LEAD EXPOSURE DATASET: IEUBK MODELING AND EVALUATION OF SOIL LEAD AS A RISK FACTOR

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1/6/95

1.0 EXECUTIVE SUMMARY

The Draft Madison County Lead Exposure Study, Granite City, Illinois, (Illinois Dept. of Public Health; Institute for Evaluating Health Risks, February, 1994) represents an evaluation of blood lead concentrations in children who live in the vicinity of the former secondary lead smelter in Granite City. The study investigated the relationships between environmental/socioeconomic/behavioral factors and blood lead.

The draft report points out that blood lead concentrations in young children were not substantially impacted, with most of the children (total of 490 6 month to 6 year old children surveyed) having blood lead concentrations well below the USEPA level of concern (10 μg/dℓ). Additionally, blood lead concentrations were dependent upon a variety of factors, with the dependence upon soil lead apparently less significant than the dependence upon factors governing exposure to lead in house paint (e.g., paint lead XRF concentrations, building condition). A follow-up critique of the draft report by USEPA challenges these conclusions, and utilizes the Integrated Exposure Uptake Biokinetic (IEUBK) Model to justify a soil cleanup goal for Granite City of 400 to 500 ppm (Marcus, 1994).

The objectives of the current analysis are presented below together with a brief summary of our major findings. The detailed analysis supporting these findings is provided in subsequent sections.

Objective #1: Review and comment upon the results of the Draft Madison County Lead Exposure Study.

The blood lead distribution shown for Granite City children is typical of that expected for urban areas. While statistical analyses of the environmental lead/blood lead relationship are confounded by a variety of covariant parameters, soil lead is unlikely to be a major explanation for elevation in blood lead.

Objective #2: Evaluation of the accuracy of IEUBK Model predictions for Granite City.

The default model used by EPA to derive a soil lead cleanup goal (Marcus, 1994) is not predictive for the cases in which soil lead exceeds 500 ppm. The slope between

blood lead and soil lead ($\mu g/dl$ change in blood lead per 1000 ppm change in soil lead) is overpredicted by approximately 4 fold by the default model.

Objective #3: Calibration of the IEUBK Model to better predict childhood blood lead concentrations at Granite City.

The best performance of the model is attained by decreasing the soil/house dust lead uptake (absorption) coefficient under conditions of high environmental concentrations. This calibration yields model predictions of the blood lead/soil lead slope that provide a good fit to the Granite City dataset. Note that TRC did not examine the algorithms embodied in the model and expresses no opinion as to their applicability in general.

Objective #4: Utilize the calibrated model to evaluate soil cleanup scenarios in terms of the possible benefits to community blood lead.

The calibrated model demonstrates that soil lead remediation to even very low concentrations (e.g., 200 ppm) would have only a slight impact upon blood lead as indicated by the limited effect of soil lead on indoor dust lead. Many children who have elevated blood lead do not live in elevated soil lead areas. The blood lead/soil lead slope factor relating blood lead to soil lead is thus shallow. Rather than focusing upon soil lead mitigation, a combined approach involving parental education, mitigation of strong lead sources (such as lead paint in poor condition, and grossly elevated soil and dust lead concentrations) may be the most effective approach, if it is decided that an intervention program of any kind is needed in this community.

Objective 5: Provide comments on the EPA critique of the Madison County draft report (Marcus, 1994).

EPA's critique focused upon a spatial relationship between blood lead and soil lead which is confounded by a variety of covariates. In the first instance, EPA appears to misinterpret the use of spatial correlation in the study. It was not intended as a method for comparing areas. Rather, it was simply intended to assure that a representative sample was obtained across the whole of the community. In any case, while soil lead and blood lead both decrease with increasing distance from the former smelter, the association between soil lead and elevated blood lead is weak and not statistically significant. Other environmental (particularly paint lead) and behavioral/ socioeconomic factors are likely stronger influences in creating blood lead exceedances. Further, EPA's use of the default IEUBK Model, which ignores the real data gathered at the site in contravention of the instructions stated in the user manual, has numerous flaws and provides a misleading assessment of the potential benefits of soil lead remediation. We believe that the cleanup scenarios presented in this analysis provide a more realistic representation of the effects of soil lead remediation.

2.0 REVIEW OF THE DRAFT MADISON COUNTY LEAD EXPOSURE STUDY

The draft report represents an evaluation of the relationship between environmental lead and blood lead levels in the community immediately surrounding the former smelter. The investigators obtained data on lead in soil, house dust, indoor and outdoor paint, and drinking water, and related these data to blood lead levels in 490 0 to 6 year old children. Smaller numbers of participants were included in the 6 to 15 year old and greater than 15 year old age groups. Additional factors considered for possible impact on childhood blood lead included: parental education and income level (socioeconomic status or SES), household number of cigarettes smoked per day, proximity to the former smelter, and age of the residence and its condition with respect to intactness of painted surfaces. Since a suitably matched control group was not identified, the study adopted a cross-sectional design relying upon regression analysis to test hypotheses regarding environmental lead: blood lead relationships. The study region was divided into concentric rings spreading outward from the former smelter to ensure a reasonably even spatial distribution of subjects, a point misunderstood by EPA.

The report provides important data on the blood lead distribution in the vicinity of the former smelter. For 0 to 6 year old children, the geometric mean blood lead was 5.58 $\mu g/d\ell$, with 16% of children having blood lead levels greater than 10 $\mu g/d\ell$. Only 7% of blood leads were above 15 $\mu g/d\ell$ in this age group. Co-linearity was found between key environmental risk factors such that soil lead, house dust lead, indoor/outdoor paint lead, condition of residence, parental income and educational level, and proximity to the former smelter site were correlated to one another. Therefore, the soil lead/blood lead relationship was confounded by a large number of interrelated variables. When hierarchical regression was used to account for key interrelated parameters (i.e., water lead, paint lead, condition of paint), it was shown that soil lead accounted for only 3% of the blood lead variance. In relation to other risk factors, the contribution of soil lead was considered to be quite small. For example, comparison of blood lead results across the soil lead <500 ppm vs. >500 ppm groups found only 1.4 $\mu g/d\ell$ differential. In contrast, a marked blood lead differential was found across residences representing different levels of upkeep. Blood lead in 0 to 6 year old children ranged from 6 $\mu g/d\ell$ when the residence was in good condition, to 8.2 $\mu g/d\ell$ for a rating of fair condition, to 11.8 $\mu g/d\ell$ for poor condition. Such findings lead to the conclusion that in this community, factors other than lead in soil have a more important impact on blood lead, in spite of the fact that soil lead levels ranged up to 3,000 ppm. Consistent with this is the results of an educational intervention in this community in which a marked blood lead decline was attributed to this intervention by the study authors.

Additional support for the concept that soil lead is not a key determinant of blood lead comes from the analyses provided below (Section 5, Figure 3) which show that the majority of blood lead exceedances in this community are in cases where soil lead is low (less than 500 ppm).

Since the Madison County Lead Exposure Study found that 16% of the 490 blood lead concentrations were above 10 $\mu g/d\ell$, there is a suggestion of a slight increase in community lead risk. The current USEPA criteria states that no more than 5% of children should be above 10 $\mu g/d\ell$.

While these blood lead data are a potential concern, it should be noted that the population geometric mean blood lead is not elevated (5.58 $\mu g/d\ell$ for 0 to 6 year old children), signifying that most children have normal blood lead. Additionally, the Granite City results are not materially different from what is found at urban areas where there is no former or current lead smelter. For example, the NHANES III dataset as compiled by Brody, et al. (1994), indicates that 16.4% of childhood (1 to 5 years old) blood lead values exceed 10 $\mu g/d\ell$ in urban areas of less than one million in population. This correspondence with Granite City blood lead results is striking and suggests that if a problem does exist at Granite City, it is best attributed to the same types of lead source that are typical of the urban environment (e.g., old housing containing dilapidated lead paint; historic lead fallout from fuel combustion). It should be noted that in the Granite City dataset, race had very little impact on blood lead, with the mean for white and non-white children not being statistically different. This contrasts with the NHANES III dataset where urban non-whites had substantially higher blood lead than did urban whites. It is possible that this indicates similar SES status for Granite City whites and non-whites since at Granite City, SES was a key determinant of blood lead.

3.0 ACCURACY OF THE DEFAULT IEUBK MODEL FOR PREDICTING GRANITE CITY BLOOD LEAD CONCENTRATIONS

Version 0.99d of the IEUBK Model was used with default parameter values (except for soil, dust and water lead where actual values were used) to provide predictions of childhood (0 thru 6 years old) blood lead levels. The model was run in the batch mode such that each record in the dataset could be put through the model and contribute individually to the overall statistics. Table 1 compares predicted and actual blood leads for the entire dataset encompassing 490 young children, and for subdivisions of the dataset based upon soil lead cutpoints. The model provided a reasonable fit to actual blood lead data for the entire dataset, both in terms of geometric mean blood lead and % greater than 10 $\mu g/d\ell$ (see top line: Total Population). However, the model overpredicted blood lead concentrations by nearly 2 fold in the soil lead subgroup that was greater than 1000 ppm (7.1 actual; 13.7 predicted). Additionally, the percentage of children with blood lead above 10 $\mu g/d\ell$ was overestimated by a large factor in this subgroup. A similar situation occurred in the 501 to 1000 soil lead subgroup, although the model overprediction was not as large (37%). In the lower soil lead groupings (0 to 250 ppm, and 251 to 500 ppm), the model-predicted blood lead was reasonably close to that actually observed, although in the lowest subgrouping, the model underpredicted by 27%.

Table 1 points out a major problem with default runs of the IEUBK Model for Granite City. The model predicts a soil lead/blood lead slope of 7.48, which is far above that actually seen (1.70). This overprediction of the slope leads to the false conclusion that blood lead is very sensitive to changes in soil lead such that if soil lead were remediated, blood lead levels should fall dramatically. The Urban Soil Lead Abatement Project (Baltimore, Cincinnati, Boston) indicated that very little benefit could be found after remediation of soil lead (e.g., Weitzman, 1993), which supports the concept of a low soil lead/blood lead slope. Other investigators have found similarly shallow soil lead/blood lead slopes (Starke, 1982; Yankel, 1977; Galke, 1975; Baltrop, 1975; Bornschein, 1990; Rabinowitz, 1988). Therefore, when modeling the benefit to be expected from soil lead remediation, it is critical

that the IEUBK Model be properly calibrated. Otherwise, the blood lead response to a change in soil lead will typically be overstated.

Note that TRC did not examine the algorithms embodied in the model and expresses no opinion as to their applicability in general.

4.0 CALIBRATION OF THE IEUBK MODEL FOR GRANITE CITY

The trend in Table 1 is that at low soil and dust lead concentrations (i.e., below 500 ppm), the model provides a good estimation of childhood blood lead. However, with increasing soil/dust lead concentration above 500 ppm, the model becomes increasingly overpredictive, such that for a significant percentage of young children at Granite City (29%), the default version of the model is inappropriate. The factor(s) which create serious model overprediction beginning at 500 ppm are not well defined, but it is clear that the model needs to be adjusted downward (i.e., less lead exposure and accumulation in blood) at the higher values for soil and dust lead. The most likely explanation for the overprediction may be decreased absorption of lead from soil and dust at higher lead loadings. This concept is consistent with a variety of literature sources (e.g., Sherlock, 1986; Bushnell, 1983) and is more plausible than other potential explanations (children contact less soil or house dust if it contains high lead; shift in lead internal distribution away from blood at higher intake).

On this basis, the model was fitted to the actual blood lead data by adjusting the model default soil/dust lead absorption coefficient (30% - total of saturable and non-saturable) to values that provide the best prediction of blood lead. The model was iterated using different absorption coefficients until a good fit was achieved for each soil lead subgrouping. Table 2 shows the back-fitted absorption coefficients that provide the best fit for several soil lead subgroupings. While the model default value of 30% soil absorption is appropriate for the 251-500 ppm group, lower absorption coefficients are required for fitting the model to actual data in higher soil lead groupings. The relationship between absorption coefficient and the composite soil/dust lead concentration approximates a straight line with a negative slope (lower absorption coefficient at higher soil leads) between 393 and 1213 ppm having a negative slope (Figure 1). This relationship can be used to approximate the lead absorption coefficient for any values of soil and dust lead in this community.

As shown in the next section, the calibrated model provided blood lead/soil lead slopes for cleanup scenarios that are reasonable estimates for that actually found at Granite City.

5.0 SOIL LEAD CLEANUP SCENARIOS

Table 3 utilizes the calibrated model to predict the blood lead benefit from remediating soil lead to 1000, 500, or 200 ppm in this community. All cleanup scenarios were run with actual batch file data for each household. Interior dust lead was adjusted to account for the remediation of soil lead by decreasing the dust lead by 0.7 times the decrement in soil lead. This approach is based upon USEPA's conservative assumption that 70% of the soil lead concentration is transferred to the indoor

environment to create house dust lead (USEPA, 1994). Our approach allows for house dust concentrations to exceed soil lead concentrations as is often the case at Granite City. The likely explanation for this differential is that interior lead sources (i.e., flaking interior paint) are a key source of dust lead. Thus, when soil lead is abated and nothing is done about interior lead paint sources, the house dust lead concentration will change by only that fraction contributed by soil lead. By adjusting dust lead by 0.7 times the decrease in soil lead, we are being faithful to the USEPA default for soil lead contribution to house dust while not ignoring other factors which contribute to house dust.

The data in Table 3 show the calibrated Model predictions of the benefit which could be expected from different soil lead cleanup targets (1000 ppm, 500 ppm, or 200 ppm). These data shown represent only the households which would be remediated, and do not take into account the overall impact on the community. This overall impact is presented in Table 4.

Table 3 indicates a modest decline in blood lead when soil leads are reduced to the indicated cleanup targets. The benefit of soil remediation is predicted to be greater above 1000 ppm (1.3 $\mu g/dl$ drop in geometric mean blood lead and 4 fold drop in percent of children with elevated blood lead) than below 1000 ppm, partially because the change in soil and house dust is larger at the higher starting concentrations. Additionally, as soil/house dust concentrations are lowered, the absorption coefficient is expected to increase thus decreasing the net effect on lead uptake. Thus, the table shows that diminishing returns are achieved by driving soil lead concentrations below 1000 ppm. It is important to note that even when soil lead is remediated to 200 ppm, the model predicts that the percentage of children with blood lead exceedances (i.e., >10 $\mu g/dl$) would still be high in the subgroup of houses that were remediated (13% above 10 $\mu g/dl$ in the 361 homes remediated to 200 ppm).

Table 4 indicates the blood lead benefit to be expected from the same three soil cleanup scenarios described in Table 3, but now data for remediated homes has been merged with the non-remediated homes (those below the remediation cutpoint) to determine the influence of remediation on the overall population (N=490 cases)blood lead. The influence of soil lead remediation on the overall population geometric mean is predicted to be miniscule, which is consistent with the fact that these cleanups would accomplish very minor reductions in population geometric mean soil and dust leads. Since the vast majority of households have soil and dust concentrations below 500 ppm, remediation of relatively few households at the top of the distribution shifts the overall exposure concentration little. Most importantly, soil remediation to concentrations as low as 200 ppm is modeled to produce only a small effect on the percentage of children with a blood lead in excess of 10 $\mu g/dl$.

It should be noted that these model simulations of cleanup scenarios yield a blood lead/soil lead slope ranging from 1.7 (Table 3) to 3.1 (Table 4). This is similar to the slope found in the current Granite City database (approximately 1.7) (Table 1). This demonstrates that the calibrated model is responsive to the actual blood lead/soil lead relationship and is likely to provide a good simulation of cleanup efficiency.

Figure 2 provides a frequency distribution of blood lead concentrations for the entire dataset of 490 cases. The observed line presents the data reported in the Madison County Lead Exposure Study. The predicted line for the 500 ppm (Pre500) cleanup scenario indicates a slight shift towards more children with blood lead values below 5 $\mu g/d\ell$, but with very little impact above 10 $\mu g/d\ell$. The 200 ppm remediation scenario (Pre200) indicates a larger shift, but with still a considerable number of children over 10 $\mu g/d\ell$.

The finding that soil lead remediation would have little beneficial effect on the community-wide rate of elevated blood lead reflects the fact that many children have elevated blood lead in spite of being surrounded by relatively low soil lead (below 500 ppm) (Figure 3). Remediation to 500 ppm will not affect these children, and it is unlikely that even lower soil remediation standards would be effective. For example, soil remediation will have little impact at homes whose house dust concentration clearly exceeds the soil lead concentration. Interior sources (e.g., paint lead) likely outweigh soil lead in such cases. The database contains 120 cases where house dust lead exceeds soil lead by 200 ppm or more, with 83 of these cases having at least a 500 ppm differential. Blood lead exceedances are a common occurrence in these cases (23%), and these cases will not be materially improved by soil lead remediation. Thus, based upon the blood lead exceedances distribution shown in Figure 3 and runs of the calibrated IEUBK Model (Tables 3 and 4), it is evident that soil lead remediation would be generally ineffectual in this community. A combined approach involving parental education, mitigation of strong lead sources (lead paint in poor condition; grossly elevated soil and dust lead concentrations) may be the most effective approach if it is decided that an intervention program is needed in this community.

6.0 EVALUATION OF EPA COMMENTS (MARCUS, 1994) ON THE MADISON COUNTY LEAD EXPOSURE STUDY

Mitigation of lead exposures is a worthwhile public health endeavor when properly directed towards high risk individuals or groups, and at the major causative factors contributing to blood lead (CDC, 1985). As pointed out above, the community as a whole does not appear to represent a high risk group for which lead mitigation would be especially required. The Madison County Lead Exposure Study and EPA's analysis suggest that subgroupings of this population may be at elevated risk in a manner which correlates with distance from the former smelter. The Madison County Lead Exposure Study points out the numerous confounding factors which affect the relationship between distance from the smelter and blood lead (e.g., year residence built, building condition, household income and education level, home ownership, soil lead). For each of these factors, residents were at greater risk (e.g., poorer building condition, lower family income and education, higher soil lead) as distance to the former smelter decreased. EPA attempts to show the relative importance of various lead sources to blood lead via correlational analyses involving spatial considerations (distance from smelter). Additionally, EPA provides a preliminary assessment of soil remediation options via the IEUBK model. However, EPA's assessment does not clearly differentiate between lead sources and provides no indication of their quantitative importance (e.g., soil lead/blood lead slope). Additionally, EPA's use of the IEUBK Model is flawed by arbitrarily assigning a default value for house dust when actual, site-specific house dust data are available. Further, the IEUBK

Model performs poorly for a large percentage of cases when model defaults (as used by EPA) are incorporated.

6.1 Assessment of Distance Rings

EPA suggests that soil lead is a more important contributor to blood lead than is paint lead based upon a distance ring subgrouping of the database. The Madison County Lead Exposure Study provided a parameter describing distance of each household from the former smelter. EPA correlated distance from the smelter with a variety of parameters which might impact blood lead to determine which factors seem most consistently associated with blood lead. Since paint lead concentrations don't vary with distance while soil lead, dust lead, and blood lead concentrations are correlated with distance, EPA suggests that soil lead rather than paint lead is the key contributor to dust and blood lead.

This correlational analysis, which focuses upon distance from the former smelter, is confounded by a variety of factors. The Madison County Lead Exposure Study demonstrates that such factors as year residence built, building condition, household income and education level, and home ownership confound the relationship between soil lead and blood lead as judged by distance. In fact, several of these factors would suggest that paint lead could become a stronger source of lead closer to the former smelter, in spite of the fact that paint lead levels are not actually correlated with distance. The fact that building condition worsens with proximity to the smelter suggests that paint lead would be more available to young children in homes nearer to the former smelter. Socioeconomic factors which affect children's exposure to lead paint sources (parental income, parental education, number of children per household) are all adversely affected with increasing proximity to the former smelter. Thus, the degree of parental supervision and awareness needed to prevent children's interaction with paint lead sources (e.g., gnawing on painted surfaces) appears to decline near the smelter. This concept is supported by the finding that children's mouthing of non-food objects increases in homes located near the smelter. These factors indicate that although paint lead levels are not correlated with distance, the degree of lead uptake from paint sources would still be expected to increase with increasing proximity to the former smelter. The likelihood that paint lead is substantially contributing to the blood lead vs. distance correlation is not recognized by EPA.

TRC examined the database subdivided by distance rings, to determine the strength of the association between soil lead and blood lead exceedances. If soil lead is an important causative factor in elevating children's blood lead above 10 $\mu g/dl$, one would expect that the frequency of blood lead exceedances at various distance rings would parallel the soil lead distribution across these rings. However, Figure 4 shows that this is not the case. This figure represents the data for distance rings 1 thru 9; distance ring 10 is not included because of the low number of cases (N=3), and distance rings 1 and 2 are combined because the number of cases in each group are relatively small (9 and 13, respectively), and because the soil and dust lead concentrations in these groups were nearly equal. It is clear from the figure that soil lead is not a major factor in elevating children's blood lead within these rings since only small changes in percent blood lead exceedances are seen (27% exceedance falls to 19%) over a soil lead range of 1000 ppm. In fact, regression of percent

blood lead exceedances in rings against the corresponding soil lead levels is not significant (p=.118; Figure 5).

In total, analysis of the dataset according to distance rings indicates a general tendency for soil lead and blood lead to decrease with increasing distance from the former smelter. However, the association between soil lead and the incidence of children with elevated blood lead is weak and not statistically significant. Other environmental (e.g., paint lead) and behavioral/socioeconomic factors are likely stronger influences in creating blood lead exceedances.

6.2 Critique of Soil Lead Remediation Goals Developed with the Default IEUBK Model

The EPA analysis concluded by back-calculating soil remediation goals based upon the default IEUBK Model. The major assumptions present in this exercise are analyzed below:

- House dust lead concentrations are 70% of soil lead concentrations, regardless of the actual house dust lead concentrations found in Granite City. EPA assumes that a soil lead-to-dust coefficient of 0.7 is appropriate because it obtained reasonable IEUBK batch mode predictions for Granite City children with this assumption. This validation exercise is flawed because it ignores the actual Granite City house dust lead data, and instead uses an incorrect default assumption. In fact, when the default model is run with the actual house dust lead concentrations, we find that in many cases (i.e., those over 500 ppm) it overpredicts blood lead concentrations (Table 1). By applying model defaults for house dust that are lower than the actual house dust data, EPA produces a reasonable fit, but one that has no basis in reality or scientific principles.

  The assumption that the Granite City environmental lead data can be described simplistically as house dust lead being 70% of soil lead is a significant error. (Note: The model's default contribution for airborne lead-to-house dust is insignificant). As the enclosed Table 1 shows, soil lead and house dust geometric means are overall, very similar. Thus, there is no basis to assume that dust lead is only 70% of soil lead. In fact, in numerous individual cases, dust lead levels far exceed the corresponding soil lead levels. Thus, while soil lead may influence house dust lead, other interior sources (e.g. lead paint) also play a fundamental role in driving dust lead. The model's remediation back-calculation option used by EPA does not allow the input of batch data files. Thus, household-specific data are lost in this exercise and an overly simplistic and incorrect model specification (house dust lead is 70% of soil lead with no significant interior sources) is introduced. This creates overestimates of the effectiveness of soil lead remediation because it ignores interior lead sources which are not affected by soil lead remediation. In other words, the assumption is that soil lead is fully responsible for house dust lead such that a 50% reduction in soil lead would yield essentially a 50% reduction in house dust lead. Obviously, if paint lead is the major contributor to house dust lead in particular homes, soil lead cleanup would have little impact on house dust or blood lead concentrations. Given the magnitude of the lead paint problem in Granite City and the number of cases in which house dust lead exceeds soil lead, the benefit of soil lead

remediation is substantially overpredicted by EPA's default back-calculation approach. This approach overlooks the well-known impact of paint lead on house dust lead. For example, Clark et al. (1985), have shown that housing stocks unlikely to contain lead paint can have low soil lead concentrations (average of 350 ppm found). However, in older homes, dust lead concentrations averaged 1,410 ppm and were two-fold higher if the home's condition was dilapidated. The USEPA Lead Criteria Document (1986) states that lead paint can be expected to substantially elevate dust lead from baseline levels.

We utilized a site-specific, non-default approach to estimate the benefit of soil lead remediation (Section 5.0). In this approach, the data from each household was run through IEUBK Model (batch mode) cleanup scenarios. It was conservatively assumed that 70% of the soil lead concentration is contributed to house dust lead, and that there are other interior sources that provide the remainder of the actual house dust lead measured. In our approach, soil lead remediation was modeled to remove that fraction of house dust lead that it is theoretically responsible for, while leaving in place that contributed by other sources (e.g., lead paint). This modeling approach is consistent with the IEUBK Model Guidance (USEPA, 1994; see pages 2-40 and 2-41), in which house dust lead is shown to consist not only of lead from soil, but also of lead from airborne deposition and from other unidentified interior (e.g., lead paint) and exterior (e.g., parental occupation) sources (the B_0 term in the house dust lead equation on page 2-41).

It should be noted that if one were to properly simulate house dust lead concentrations, the model offers a Multiple Source Analysis in which interior lead sources can be factored in. By using the default in this case (no interior sources), EPA misses an opportunity to render its use of the model more realistic. However, the best approach is still to run remediation scenarios in the batch file mode in which the actual house dust lead concentrations can be adjusted downward based upon the anticipated benefit from soil remediation. This is the approach we took in Section 5.0.

- Lead bioavailability from soil does not reflect the non-linearities observed at Granite City. The IEUBK Model assumes that the ½ saturation point for soil lead absorption is 100 μg lead ingestion/day. In other words, it would require a lead loading of 200 μg/day to begin to see a curvilinear (saturation) lead uptake response. With current model defaults, this level of lead loading would require a soil/house dust concentration of approximately 2,000 ppm. As discussed in Section 4.0, non-linearities which may involve saturable uptake occur at much lower concentrations. Therefore, the default version of the model is insensitive to the non-linearities seen at Granite City, and it must be modified if it is to be predictive of the benefits of soil lead remediation. The modifications described in Section 4.0 address these non-linearities, while the default approach used by EPA does not.

- A soil lead/blood lead slope of 7 to 8 μg/dl blood lead change per 1,000 ppm soil lead change. The model default creates this large slope by underestimating the non-linearities in the lead absorption profile, and by ignoring other factors which continue to add to blood lead

(e.g., paint lead, other lead sources) even when the soil lead concentration has been reduced. Thus, the benefit of soil lead remediation is grossly overpredicted by the default version of the model.

Our model runs, calibrated for the Granite City dataset using actual environmental data, provide a much more realistic soil lead/blood lead slope. The analysis provided by EPA is not site-specific and uses assumptions which overemphasize the influence of soil lead on blood lead. EPA's use of a default model that overpredicts the soil lead/blood lead slope by 4-fold (Table 1) indicates that the default model does not apply to Granite City. Our use of each household's data in validation runs and soil lead remediation scenarios provides a major advance over the default approach used by EPA.

7.0 REFERENCES

Baltrop, D.; Strehlow, C.D.; Thornton. I.; Webb. J.S. (1975) Absorption of lead from dust and soil. Postgrad. Med. J. 51: 801-804.

Bornschein. R.; Clark, S.; Pan. W.; Succop, P. (1990) Midvale community lead study, Final Report.

Bushnell, P.J.; DeLuca, H.F. (1983) The effects of lactose on the absorption and retention of dietary lead. J. Nutr. 113: 365-378.

Centers for Disease Control (CDC) (1985) Preventing Lead Poisoning in Young Children.

Clarke, C.S.; Bornschein. R.L.; Succop, P.; Que Hee, S.S.; Hamond, P.B.; Peace, B. (1985) Condition and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. Environ. Res. 38: 46-53.

Galke, W.A.; Hammer, D.I.; Keil, J.E.; Lawrence, S.W. (1975) Environmental determinants of lead burdens in children. In: Hutchinson, T.C.; Epstein, S.; Page, Al. L.; Van Loon. J.; Davey, T.; eds. International conference on heavy metals in the environment: symposium proceedings, Vol. 3, October: Toronto, ON, Canada. Toronto, ON Canada: Institute of Environmental Studies, pp. 53-74.

Marcus, A.H. (1994) Preliminary Assessment of Data from the Madison County Lead Study and Implications for Remediation of Lead-Contaminated Soil.

Rabinowitz, M.B.; Bellinger, D.C. (1988) Soil lead-blood lead relationship among Boston children. Bull. Environ. Contam. Toxicol. 41: 791-797.

Sherlock, J.C.; Quinn, M.J. (1986) Relationship between blood lead concentrations and dietary lead intake in infants: the Glascow Duplicate Diet Society 1979-1980. Food additives and contaminants 3: 167-176.

Stark, A.D.; Quah, R.F.; Meigs, J.W.; DeLouise, E.R. (1982) The relationship of environmental lead to blood-lead levels in children. Environ. Res. 27: 372-383.

United States Environmental Protection Agency (EPA) (1986) Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children. EPA 540-R-93-081.

United States Environmental Protection Agency (EPA) (1986) Air Quality Criteria for Lead, Vol. III EPA 600/8-83/028aF-dF, Environmental Criteria and Assessment Office, U.S. Environmental Protection Agency, Research Triangle Park, NC.

Weitzmann, M.; Aschengrau, A.; Bellinger, D.; et al. (1993) Lead-contaminated soil abatement and urban children's blood lead levels. J. American Med. Assoc. 269: 1647-1654.

Yankel, A.J.; von Lindern, I.H.; Walter, S.D. (1977). The Silver Valley lead study: the relationship between childhood blood lead levels and environmental exposure. J. Air Pollut. Control Assoc. 27: 763-767.

TABLE 1
DEFAULT RUNS OF THE IEUBK MODEL

Case	N	G Mean Soil Pb	G Mean Dust Pb[a]	Actual		Predicted	
				G Mean Blood Pb	% > 10 μg/dl	G Mean Blood Pb	% > 10 μg/dl
Total population	490	335	334	5.60	16	6.13	19
Soil Pb >1000 ppm	39	1510	971	7.10	31	13.69	85
Soil Pb 501-1000 ppm	104	651	719	6.43	19	8.83	24
Soil Pb 251-500 ppm	169	347	430	5.69	20	6.21	17
Soil Pb 0-250 ppm	178	146	196	4.78	7	3.48	3
Soil lead/blood lead slope[b]				1.70		7.48	

[a] In certain cases the median dust lead is reported since a geometric mean value could not be calculated due to zero values in the dataset.

[b] Rate of change of blood lead (μg/dl) per 1000 ppm change in soil lead based upon the geometric mean soil and blood lead values shown in the table.

TABLE 2
CALIBRATION OF IEUBK MODEL ABSORPTION COEFFICIENT

Case	TWA Soil/Dust Pb[a]	Actual Blood Pb	Back-Fitted Absorption Coeff.
Soil Pb >1000	1213	7.10	12%
Soil Pb 501-1000	688	6.43	19%
Soil Pb 251-500	393	5.69	30%

[a] Time weight-averaged soil and dust lead concentration. Derived by averaging soil and dust lead concentrations according to the model default (45% exposure to soil, 55% exposure to dust).

TABLE 3
IEUBK MODEL RUNS FOR CLEAN-UP SCENARIOS
(STATISTICS FOR REMEDIATED CASES ONLY)

Clean-up Scenario	# Cases Cleaned	Pre-Clean-Up				Post-Clean-Up			
		G.M. Soil Pb	G.M. Dust Pb	G.M. Blood Pb	% >10	G.M. Soil Pb	G.M. Dust Pb	G.M. Blood Pb	% > 10
to 1000 ppm	39	1510	970	7.1	31	1000	743	5.8	8
to 500 ppm	143	819	780	6.59	22	500	596	5.95	15
to 200 ppm	361	460	411	6.0	19	200	228	5.4	13

Figure 1

Backfitted % Absorbtion vs. TWA for Pb in Soil & Dust

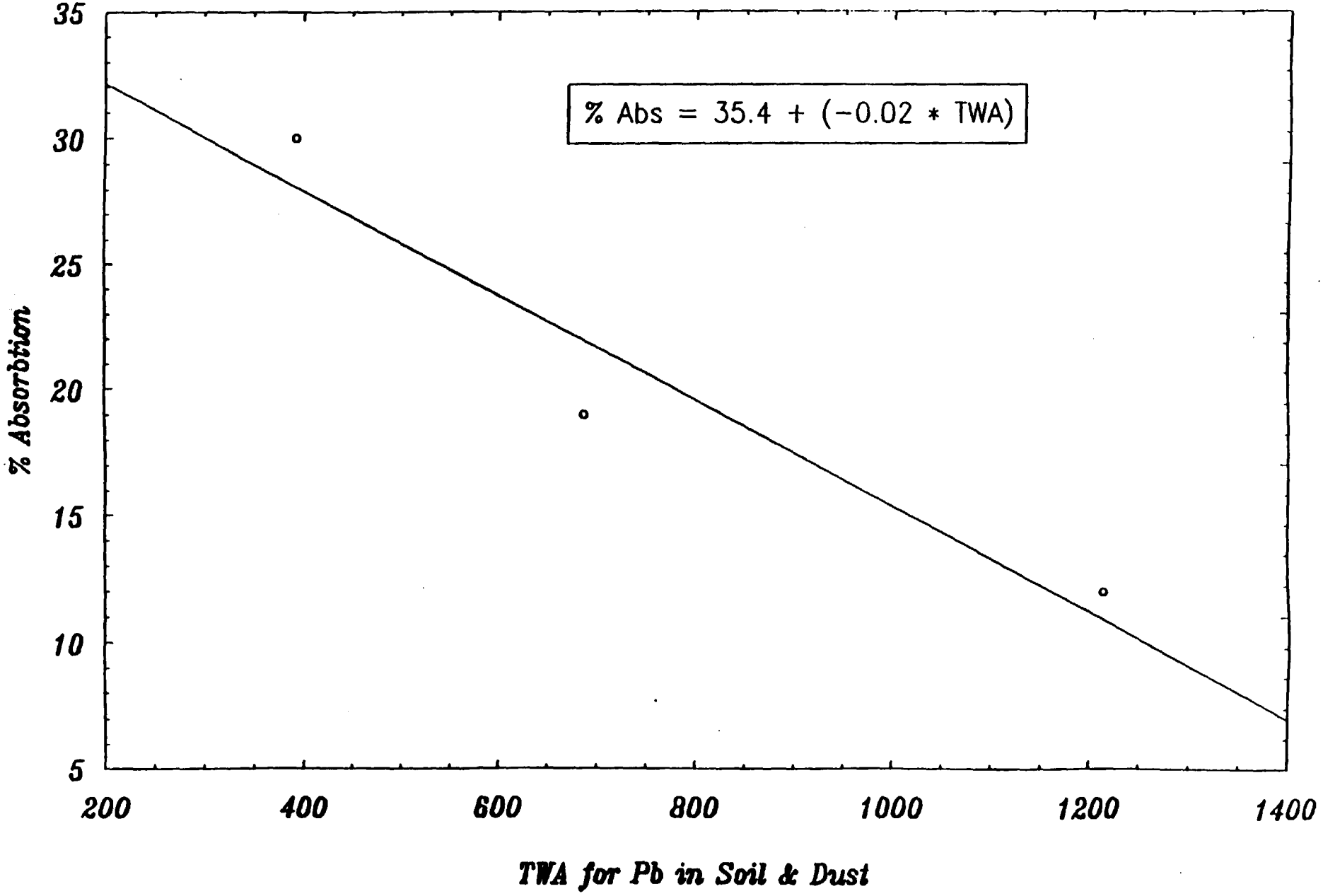

Figure 2

Frequency Plot for 200 & 500 ppm Soil Pb Cleanup

(N=490)

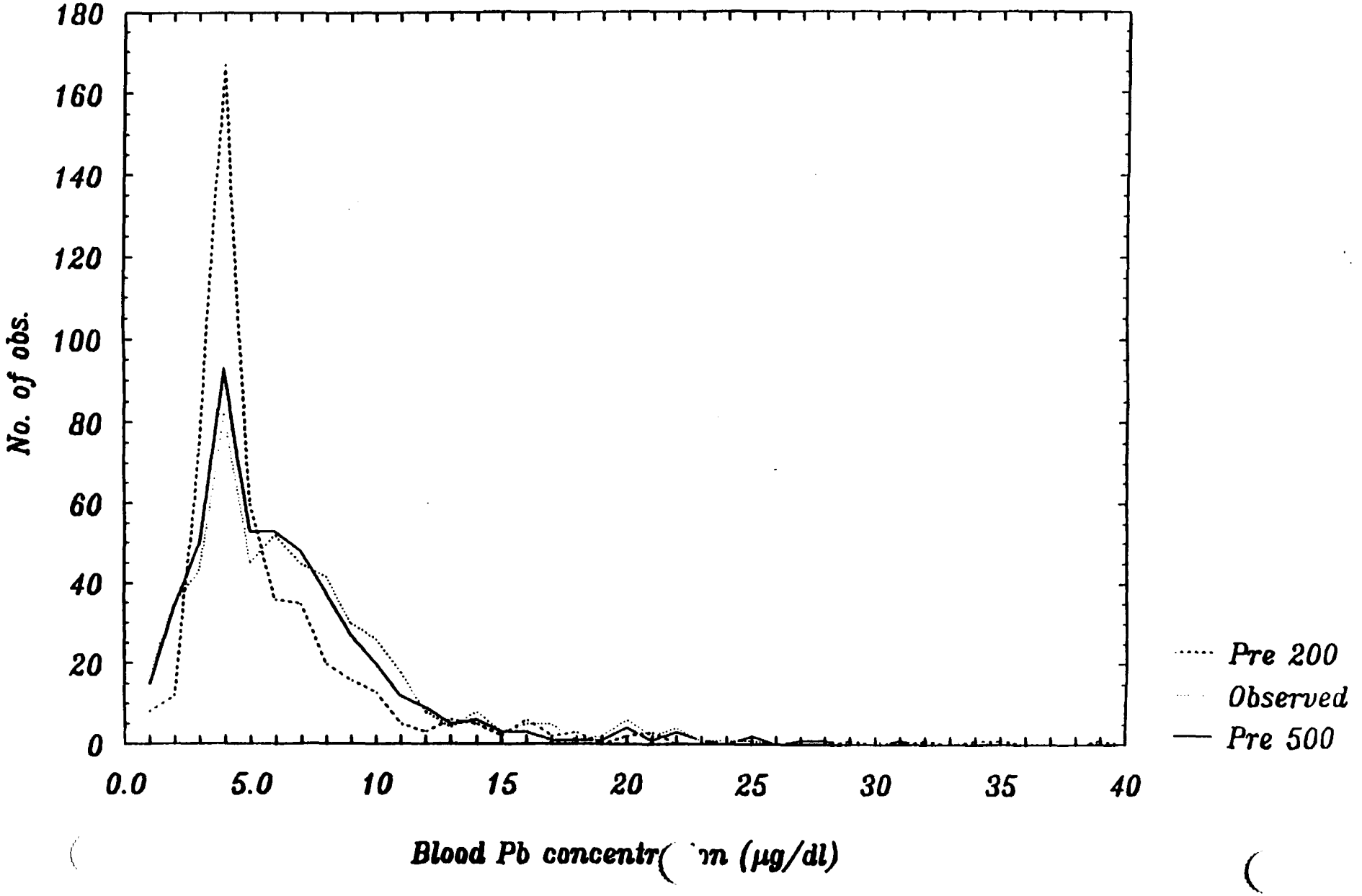

FIGURE 3

BLOOD LEAD EXCEEDANCES ACROSS SOIL LEAD SUBGROUPS

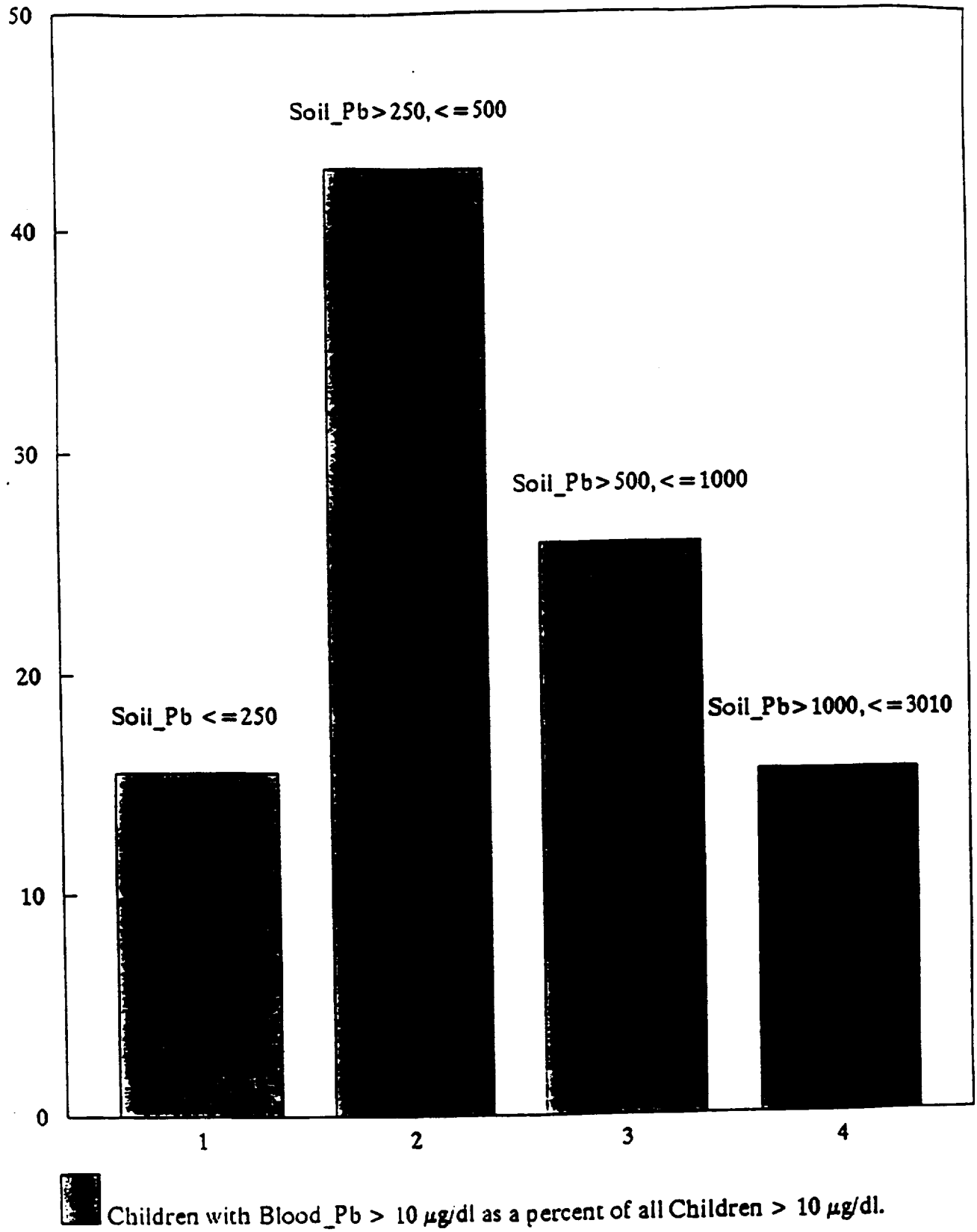

Figure 4

Comparison of Soil Pb concentration and Blood Pb exceedances over distance

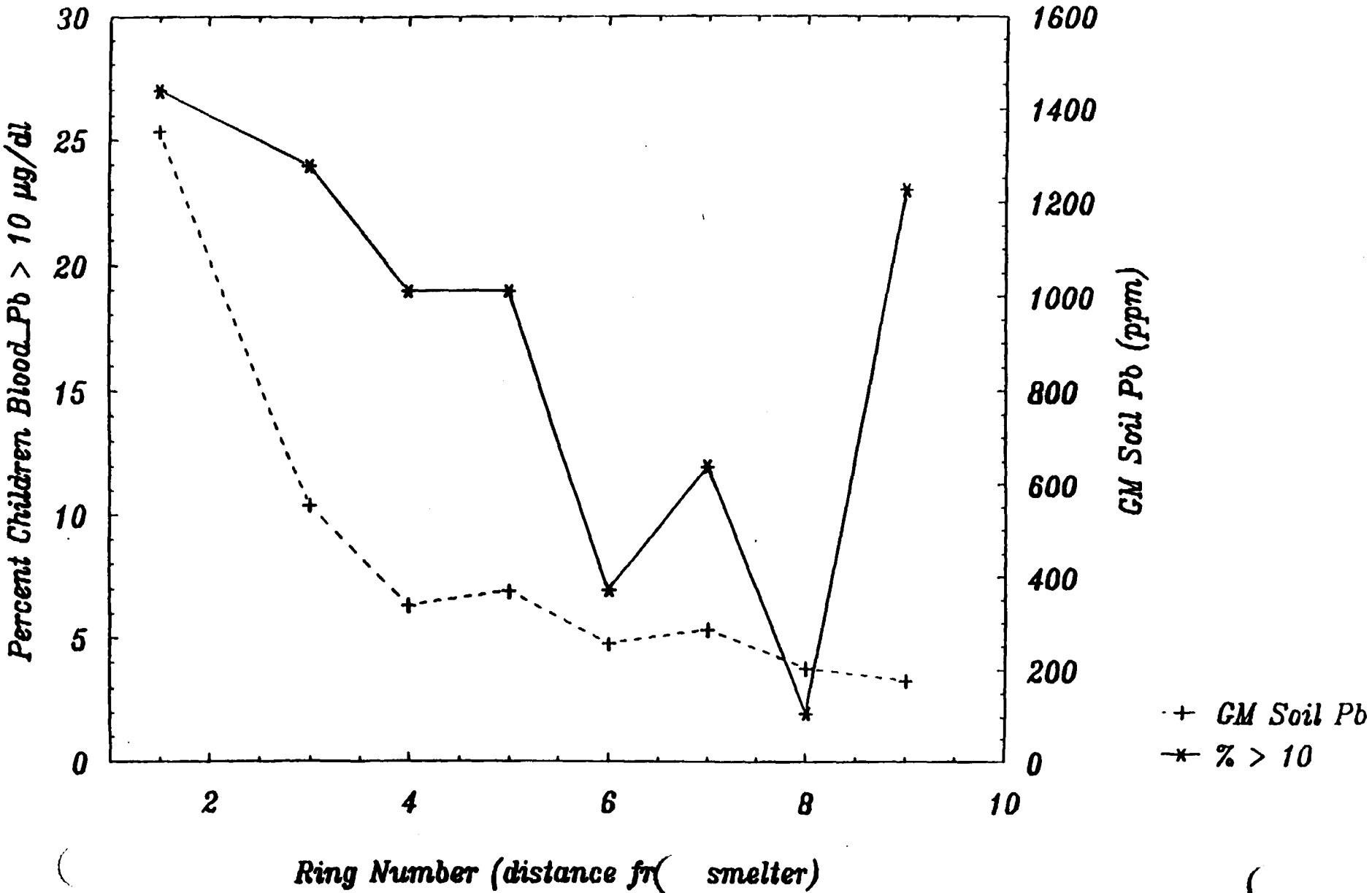

Figure 5

Regression of Blood Pb exceedances vs. Soil Pb

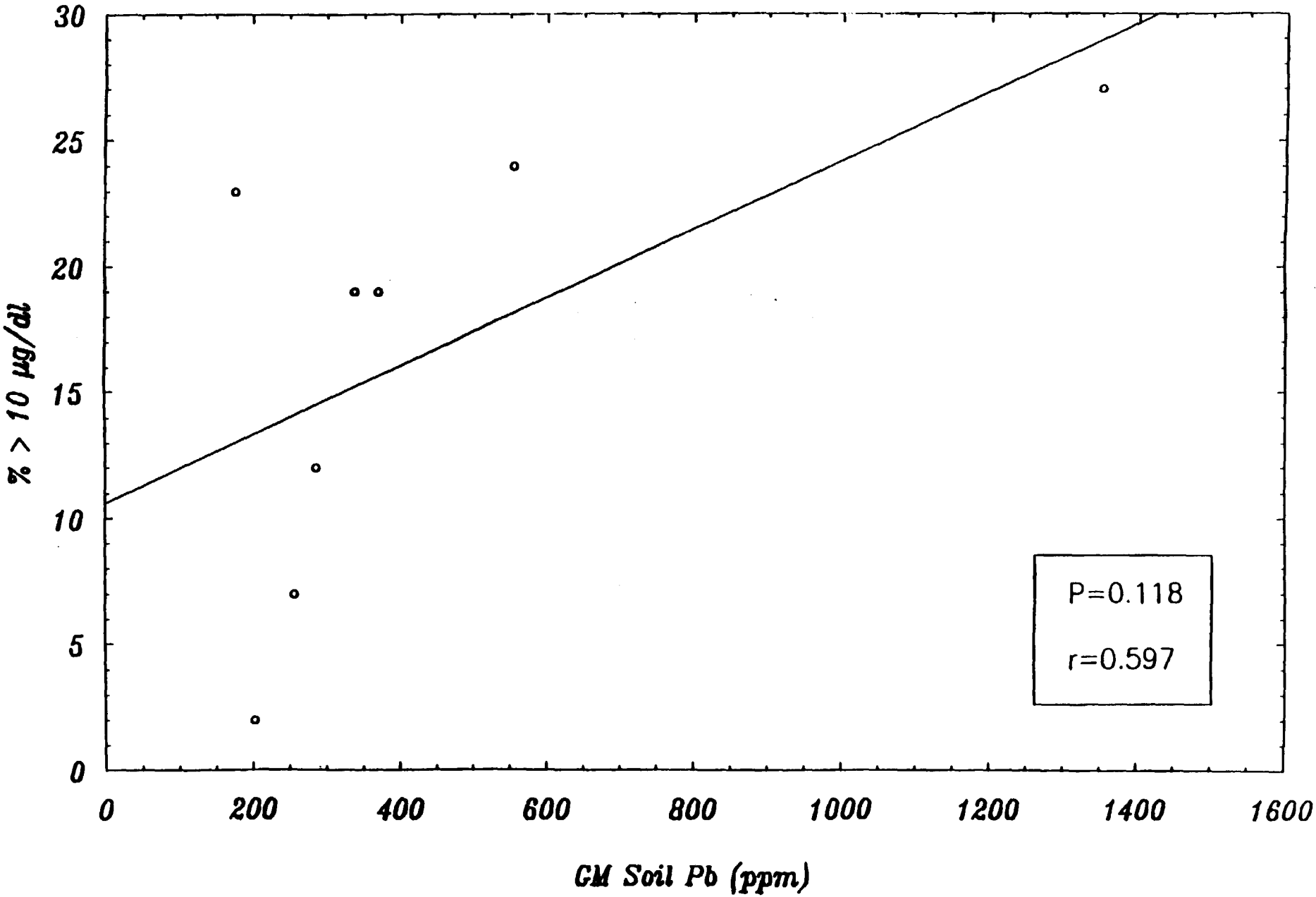

EXHIBIT A

REVIEW OF PUBLIC RECORD DOCUMENTS FOR THE NL INDUSTRIES / TARACORP SITE Granite City, Illinois

Prepared for:

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January 13, 1995

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January 13, 1995

EXECUTIVE SUMMARY

The PRP Group for the NL Industries/Taracorp Site has retained McLaren/Hart to provide a review of the documents for the NL Industries/Taracorp Superfund Site (Site) which were introduced into the Administrative Record by the United States Environmental Protection Agency (USEPA) in support of their recommended 500 ppm lead in soil cleanup level at the Site. McLaren/Hart evaluated the significance of each reference as it pertains to the establishment of the 500 ppm cleanup criteria for lead in residential soils.

No document reviewed supported or demonstrated that soil lead cleanup levels of 500 ppm would significantly reduce lead in blood. One of the documents reviewed indicated only a slight increase in PbB (1.25 μg Pb/dL of blood) was associated with a 1000 mg/kg increase in soil concentrations for an inactive smelter site based on a study conducted in Midvale, Utah. Assuming an average background PbB in a child is 5 μg/dL, the results from the Midvale study suggest that a soil concentration of 4000 mg/kg would be required to reach a blood lead concentration of 10 μg/dL. Additionally, the relationship between PbB and PbS was weak and confounded by variables such as socioeconomic factors, diet (calcium, zinc and iron), the amount of lead ingested, PbS bioavailability, and sources of lead other than PbS [air, dust, and lead-based paint (LBP)].

Overall, the documents reviewed do not support the recommendation of a 500 ppm cleanup level for lead in residential soils as a mechanism for mitigating potential health risks associated with elevated blood lead levels in children living in the vicinity of the NL Industries/Taracorp Site.

The majority of the information reviewed supports the concept that cleanup levels should be determined for each site using site-specific data along with available data analysis techniques and current toxicological information to develop an appropriate cleanup level or Preliminary Remediation Goal (PRG) based upon protecting human-health and the environment. This analysis includes the incorporation of site-specific information into the IEUBK and other computerized statistical modeling approaches. Once the proposed cleanup level has been developed, it should then be subject to evaluation by the scientific, regulatory and public communities which will be affected by its implementation. The consideration of health-related, economic, social and scientific factors must be weighed prior to finalization of any cleanup standard in order to determine the best possible remedial action for the site. Such an approach is consistent with USEPAs recent guidance for determining protective levels for lead in soil at CERCLA sites (OSWER Directive #9355.4-12: Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities).

Conclusions

- The documents placed in the Administrative Record do not support the recommendation of a 500 ppm soil clean-up level for Pb for the Site;

- Comparisons can be made with other similar sites that indicate 1000 ppm Pb or higher would be an appropriate soil cleanup level for residential areas of the Site; and

- Site-specific analyses (beyond those already conducted by USEPA) are required to develop the most appropriate clean-up level for this Site. An analysis such as the IDPH study can serve this purpose (84).

1.0 INTRODUCTION

The NL Industries/Taracorp Site (Site) occupies approximately 16 acres in Granite City, Illinois. Hoyt Metal first operated the Site for metal refining, fabricating and related work in 1903, and metal operations continued at the Site under several different owners. During this period, facility operations included a secondary lead smelter used for purifying/reprocessing lead-containing scrap and used batteries. Smelter operations ceased in 1983.

In December of 1982 the Site was proposed for the National Priorities List (NPL). NL Industries, as previous owner of the Site and one of the potentially responsible parties (PRP) voluntarily entered into an Agreement and Administrative Order by Consent (Order) with United States Environmental Protection Agency (USEPA) and the Illinois Environmental Protection Agency (IUEPA) in May of 1985. Under this Order, NL Industries was required to initiate a Remedial Investigation/Feasibility Study (RI/FS) to evaluate the nature and extent of contamination at or near the Site and assess remedial alternatives as necessary. USEPA divided the Site into three areas of concern regarding lead impacts as a potential health threat to the community: the Main Industrial Properties, the Adjacent Residential Areas, and the Remote Fill Areas.

As part of this process, the USEPA has proposed a cleanup level of 500 ppm (500 mg/kg) for lead in the Adjacent Residential Area soils (March 1990 ROD). In support of this proposed cleanup level, USEPA has introduced 115 references into the Administrative Record for the NL Industries/Taracorp Site. In addition, the USEPA has opened a Public Comment Period to address issues associated with the proposed residential soil cleanup level and subsequent remedial strategy. Public comments will be considered by the USEPA prior to finalization of the soil lead cleanup level (115).

The PRP Group has retained McLaren/Hart to provide a review of the documents introduced into the Site's Administrative Record in support of the 500 ppm lead in soil cleanup level in order to evaluate the significance of each reference as it pertains to the establishment of the 500

ppm cleanup level for lead in residential soils. McLaren/Hart was able to obtain and review all 115 documents that were introduced into the NL Industries/Taracorp Site Administrative Record on October 13, 1994. The documents submitted by USEPA do not support its position.

A general overview of McLaren/Hart's review is provided in Section 2.0 with a more detailed discussion of these issues presented in Sections 3.0 through 5.0.

2.0 GENERAL OVERVIEW

A majority of the documents placed into the Administrative Record for the NL Industries/Taracorp Site fall into one of three general categories: toxicological issues surrounding lead, statistical and modeling techniques for analysis of lead data, and issues surrounding lead-containing soils.

These three general categories presented information on the following issues:

Toxicological:

- Health effects,
- Biomarkers, and
- Metabolism/pharmacokinetics of lead (bioavailability, intake, and distribution).

Statistical/Modelling Approaches:

- IEUBK Model,
- Regression analyses, and
- Structural equations.

Lead-containing Soils:

- Multi-media issues,
- Exposure pathways,
- Environmental predictors of blood lead concentrations,
- Site-specific case studies, and
- Development of cleanup criteria for lead in soil.

The review process revealed that several of the supporting references included duplication of various journal articles or study data. In the event of duplication resulting from the inclusion of draft and final versions of reports or publications presented within more than one reference, only the final draft or most recent of the reference was reviewed.

In addition to duplication of references, McLaren/Hart found that several of the cited references were quite dated (as far back as 1961). Information contained in these articles are not the most appropriate for supporting the development of current cleanup levels based upon the myriad of changes that regulatory guidance has undergone in recent years in response to the increased awareness and evidence regarding the potential health effects of lead exposure.

3.0 DETAILS OF TOXICOLOGICAL ISSUES

Health Effects

Of the documents reviewed, approximately 70 addressed toxicological issues. These documents addressed health affects and characterized human lead toxicity from a variety of multi-media sources including environmental media, gasoline, food, soldered cans, leaded ceramic ware and crystal decanters, and occupational sources. In general, the documents noted that sources of lead from gasoline, soldered cans, food (dietary), lead-based paint (LBP), and air were the greatest sources of human exposure to lead (6, 15, 18, 31, 35, 36, 39, 77, 81, 83, 86, 109, 111, 113). Environmental media sources were predominately grouped as air, water, dust, soil, and paint, and **only 10 of the 70 articles on toxicological issues individually evaluated lead in soil (PbS) as a source.** A review of the articles relating to such health effects is not included in this document.

Biomarkers

Several documents submitted to the record reviewed biomarkers for determining long-term lead exposure; such biomarkers include bone, teeth, nails and hair (36, 55, 86). However, the long-term biomarker studies reviewed did not specify soil as the primary source of lead, and conflicting results were reported for correlations of lead effects and long-term biomarkers. This suggests that a strong relationship between long-term biomarkers and lead toxicity was not well established, even though a correlation of long-term biomarkers to PbB may exist (58).

In one study, children with high tooth lead concentrations (18 μg/g) were reported to have lower visual motor skills but not lower intelligence scores (52), while deficiencies in intelligence, attention span, auditory language, function and disordered class behavior were reported in two similar studies (19, 23). However, one of the researchers was investigated for misuse and manipulation of study data which suggests that these conclusions should be viewed with caution (98, 99). Another study reported only a small relationship between tooth lead and school performance (44). This inconsistency of lead related health related effects correlated with long-term biomarkers was reportedly due to socioeconomic factors, diet, nutrition and interpretation of behavioral characteristics, which all confound the results and make overall evaluation difficult (11, 41, 99).

From the long-term biomarker study documents reviewed, it also appeared that the use of bone, tooth, nail and hair biomarkers were not as widely used as PbB monitoring, which is a better measurement of lead toxicity in children. Instead the long-term monitors of lead were applied as an alternative monitoring technique during chelation therapy and for studying the distribution and deposition of lead in the child and adult skeletal system (9, 33, 47, 49, 60, 70). Specifically, the bone lead studies noted that (1) concentrations of bone lead increase with age, (2) the turn over of bone lead was relatively slow, and (3) children have less bone lead because they have less dense bone where lead is primarily stored (6, 8, 10, 16, 17, 68, 69). This reduced tendency for children to store lead in bone may limit monitoring low level lead exposures and the identification of related health affects.

When identifying the specific source of lead exposure in infants, children and adults, the documents reviewed cited leaded gasoline, LBP and food as the primary source of lead intake and lead toxicity (18, 81, 111, 113). Historically a reduction in PbB concentrations of 80% was observed due to the elimination of lead soldered cans (113). Similar reductions were reported in another study that measured a PbB decrease of 72% to 97% for the entire United States population and selected subgroups when the use of leaded gasoline and lead soldered cans was eliminated. The primary remaining sources are lead in paint, dust and soil, however, the individual effect of each of these sources is difficult to identify (111).

It is unclear why the documents on biomarkers were placed in the Administrative Record, because these documents do not support the 500 ppm proposed cleanup level. They may have been included to indicate PbB as the preferred and most easily accessible biomarker of exposure.

Bioavailability

Of the toxicological documents reviewed, several documents specifically addressed exposure to lead in soils (PbS) and the measurement of PbB to identify the lead toxicity and a relationship of PbB to PbS exposure. These documents primarily described bioavailability of PbS from mining, milling and smelter sites based on animal study data, and attempted to correlate PbB to PbS at these sites using various modeling techniques (113). The documents on bioavailability were likely placed in the Administrative Record to indicate the need to use site-specific data.

The bioavailability of PbS from mining, milling, and smelter sites was reported to be influenced by many factors. Age, nutrition, stomach pH, particle size, solubility, and lead species were all important determinants of lead intake and bioavailability (2, 32, 63, 67, 113). Adults were reported to adsorb roughly 10% to 20% of dietary lead, and infants/children 8 months to 8 years old were reported to adsorb 40% to 50% of the total dietary lead intake. However, the bioavailability of PbS was reported to be lower than the bioavailability of lead from dietary lead sources due to particle size, solubility, and lead speciation.

The reviewed documents contained bioavailability studies of PbS using animal studies and modeling studies to simulate bioavailability in mining-derived and smelter-derived PbS. In a study of Sprague-Dawley rats, the distribution of bioavailable lead from mill tailings was 20% in blood, 9% in bone, and 8% in the liver. It was also noted that the effect of PbS dose on the bioavailability of PbS was low, and a weak relationship between PbB and PbS was acknowledged (113). This finding was consistent with the results of the Butte study of lead from milling tailings where child PbB mean concentrations were 3.5 μg Pb/dL.

Similar bioavailability findings were reported in a second rat study using mill tailing particles less than 100 micrometers (μm) (80). The similarity in findings of these two studies suggests that the bioavailability of lead in mill tailing particles may not be directly related to particle size since the smaller particles would be expected to yield greater bioavailability which was not encountered. However, one study reported that the bioavailability data from rat studies was deficient due to differences in rat and child metabolism, and suggested that swine are a better model for correlating bioavailability in children. In a study of young swine, lead in mill tailings was reported to be more bioavailable (113) than was observed in rats. Using < 100 μm particles of mining tailings, this study indicated that the form of PbS (lead sulfate) had no affect on bioavailability, and the bioavailability of mining-derived PbS was equivalent to that of food (dietary).

Tracer studies and modeling studies of PbS bioavailability were also used to quantify soil intake and adsorption. Due to tracer method variability, subject-specific differences, inconsistencies, and interferences of lead from food sources, these studies were approached cautiously, and were reported to be reliable if the appropriate methodological considerations were taken and resulting

conclusions evaluated carefully (113). Modeling studies were also used to measure bioavailability The models incorporated particle size, solubility, and speciation to predict the bioavailability of PbS. The overall conclusion was that both tracer studies and modeling studies require further research and site-specific parameters to be accurate in assessing bioavailability of lead in soils.

In a study of the bioavailability of lead in mining-derived PbS, a relationship between PbB and PbS was referenced from Steel et. al., 1990, which was not included in the 115 documents reviewed. From epidemiological studies, the effect of mining-derived PbS on PbB was less than the effect of urban sites and sites with an active smelter. Specifically, an increase of PbB from 1.1 μg Pb/dL to 7.6 μg Pb/dL per 1,000 ppm PbS was calculated for an active smelter site relative to an increase in PbB of 0.0 μg Pb/dL to 4.8 μg Pb/dL per 1,000 ppm PbS for children living near mining-derived PbS sites (113). It was not noted whether or not the mining site and active smelter site PbB to PbS relationship accounted for the contributions of lead in air, dust, and paint. Therefore, these studies may have actually overstated the relationship of PbB and PbS (22, 30).

A study of the relationship between PbB and PbS was conducted for children living near an active lead smelter in Jamaica. In this study of child PbB and PbS, a PbB of 8.6 μg Pb/dL per 1,000 ppm PbS was calculated. Although this PbB increase was slightly higher than the increase found in the epidemiological study (1.1 μg Pb/dL to 7.6 μg Pb/dL PbB per 1,000 ppm PbS), this study noted that the affects of air emissions, surface dust and LBP were not considered (113). Furthermore, the researcher reported that increases in PbB from smelter-derived soils would be different from developed areas due to diet, hygiene, and climate. In Jamaica, children were likely to engage in outdoor activities throughout the year. Therefore, the PbB to PbS relationship presented would further overstate the increase in PbB from PbS (113).

A lower PbB to PbS relationship was also supported by the Midvale study where a relatively lower increase of PbB, 1.25 μg Pb/dL PbB per 1,000 ppm PbS, was modeled for children in close proximity to a mill and inactive smelter sites. The study findings noted that LBP and not PbS was the principal contributor to the increased PbB, and the contribution from LBP was correlated with a PbB increase of 4.0 μg Pb/dL (113). Additionally, only an indirect

relationship of PbB to PbS was made with respect to the inactive smelter and mill sites. The indirect correlation was based on the direct relationship of PbS concentrations which increased nearer the sites, and incorporating these soil concentrations into a model to predict PbB. In concluding, the study indicated that the low concentrations of PbB from PbS were due to (1) the decreased bioavailability of PbS and (2) dietary factors.

In sum, the toxicological documents do not support the proposed 500 ppm cleanup level. In fact, based on the Midvale Study described above (113), only a slight increase in PbB (1.25 μg Pb/dL of blood) was associated with a 1000 mg/kg increase in soil concentrations for this inactive smelter site. If you assume an average background PbB in a child is 5 μg/dL, then it would require a soil concentration of 4000 mg/kg to reach a PbB of 10 μg Pb/dL of blood. The linear relationship may or may not be valid at low soil lead concentrations. Valid kinetic modeling would be the preferred method of predicting blood lead levels resulting from these lower exposures. Additionally, the relationship between PbB and PbS from mining-derived PbS was weak and confounded by variables such as socioeconomic factors, diet (calcium, zinc and iron), the amount of lead ingested, PbS bioavailability, and sources of lead other than PbS (air, dust, and LBP).

4.0 DETAILS OF STATISTICAL/MODELLING ISSUES

A small number of the reviewed documents presented information regarding the various modeling techniques that may be utilized in assessing lead data and predicting environmental (soil, air, dust) as well as blood lead concentrations. **While these documents present useful information pertaining to the statistical analysis of lead data, they did not support the establishment of the 500 ppm soil lead cleanup standard for this Site.**

Several of the reviewed papers were authored by Allan H. Marcus, and addressed the various statistical approaches that can be employed to estimate the relative importance of different sources and pathways of childhood lead exposure. These methods include the use of structural equation modeling, physical tracers of source and mass balance equations (95).

Structural equation modeling is used in the estimation of both direct and indirect exposures to LBP and is often used in cross-sectional field studies to demonstrate the role of LBP as both a direct (ingestion of paint chips) and indirect (soil, dust air) source of lead exposure. Physical identifiers of a lead source (i.e. an unusual ratio of stable lead isotopes) provide the basis for stronger causal inferences to be drawn about sources and pathways of lead exposure (95).

Using these approaches, Marcus found that the relationship of soil to LBP is significant, estimating that an additional 230 mg/kg of lead in soil was evident at homes where exterior LBP contained concentrations of lead greater than 1 mg/cm^2. In addition, he concluded that exterior LBP, is a source of lead in soil regardless of air lead point sources being present. This soil then contributes significantly to the household dust fraction of lead providing a potential exposure for building occupants (95).

Other modeling issues discussed the use of USEPA's Integrated Exposure Uptake Biokinetic (IEUBK) computer model which is designed to model exposure from lead in air, water, soil, dust, diet and paint and other sources with pharmacokinetic modeling to predict blood lead levels in children 6 months to 7 years old (97). A detailed review of this model is being conducted by another committee in support of the public comment period review and therefore was not reviewed in detail for the generation of this document.

Additional modeling and statistical issues included the study of the relationship of PbB and lead in air. A comparison of the Goldsmith-Hexter log-log model and the linear total exposure model (C.R. Angle) suggested that the linear total exposure model should be used for determining lead concentrations in air versus the Goldsmith-Hexter log-log model because the latter of these two models does not account for a background concentration of PbB in children (1, 25). Another study of lead in air suggests that much variability is introduced into the air pathway from lead in soil, dust, LBP etc. These become confounding variables (i.e. variables that are related to both the dependent variable and independent variable) and must be considered when establishing a relationship between environmental lead sources and increases in PbB. This analysis is very complex and the researchers in this study were not able to make a reliable quantitative estimate of the relationship between lead in air and PbB in children (26).

These studies, similar to the toxicological studies that were reviewed, reveal the complexity and variability associated with the analysis of lead data. Based upon this observation, decisions regarding lead exposure and the potential remedial actions associated with reduction and/or elimination of that exposure must consider a variety of factors ranging from scientific to socioeconomic issues. It is noteworthy that the USEPA IEUBK model has never been published in the peer-reviewed scientific literature although it has undergone Science Advisory Board review. In addition, no mention was made of other peer-reviewed models that could be applied in analyzing the data from the IDHP study (96).

5.0 DETAILS OF SOIL ISSUES

When addressing issues surrounding the remediation of contaminated soils the question of "how clean is clean?" is relevant. The definition of "clean" encompasses a number of variables which differ from site to site. Consequently, cleanup levels must be developed on a site-specific basis in order to allow for remedial options which are protective of human health and the environment while remaining cost-effective. The USEPA has suggested that the point of departure for defining clean soil at residences near the NL Industries/Taracorp Site is a lead concentration of 500 ppm (500 mg/kg)(112).

For a substance to be considered hazardous, it must have the potential for producing adverse effects on the health or safety of humans or the environment. It would follow that the driving force in the development of hazardous waste cleanup levels must be the issue of adverse health effects or ecological impacts. The health effects presented by exposure to elevated concentrations of lead are more prominent in children than adults.

Multimedia Issues and Exposure Pathways

The primary sources of childhood lead exposure include LBP, urban soil and dust, drinking water, and food (2, 4, 32, 71, 72, 74). Sources of lead in soil and dust results from a combination of environmental factors including: deteriorating LBP, stationary atmospheric emission sources (such as the non-ferrous metal industry (46)), previous waste disposal practices, and automobile exhaust (56).

While the measurement of lead in blood provides a good indication of lead exposure, the variables which contribute to each exposure are much harder to define. This is because lead is a multimedia pollutant and, as such, the number of children exposed to lead in dust and soil cannot be separated from the numbers exposed to airborne lead or LBP. One reason for this is because simultaneous exposures are occurring from these media and secondly because LBP and airborne lead are primary contributors of dust and soil lead (36, 56, 86). Researchers have studied various exposure pathways in an attempt to quantify the individual contribution of multiple lead sources. Several of the papers reviewed attempted to rank exposure sources based upon their overall contribution to blood lead. Many of the results were able to draw correlations

(direct or indirect) between exposure sources and blood lead concentrations (12, 20, 29, 38, 43, 71, 72, 74, 90, 93, 95, 102, 104), but a rigid, quantifiable ranking of each source's contribution must take into account numerous confounding variables making this process complex and subject to much variation (4, 56, 75, 89).

Environmental Predictors

While the concentration of lead in soil (PbS) may contribute to the concentration of lead in house dust (PbHD) (5, 43), there are other factors which may contribute more, such as paint. There are studies that purportedly support a correlation between PbHD and blood lead concentrations (PbB) (75, 29).

In addition to lead concentrations in paint, dust and soil, there are many other factors which influence lead concentrations in blood. These include such things as socioeconomic factors (household income, parental level of education), exposure to cigarette smoke, parental occupational exposure, and minority classification (12, 21, 43, 110). The significance of such factors was identified in an evaluation of a study conducted in New Haven, CT where a low level of socioeconomic status was correlated with increased blood-lead levels (20). This type of evidence demonstrates the complex nature of identifying and quantifying exposure from individual sources of lead. **Identifying a single, predominant environmental source or pathway of lead exposure in children with blood lead concentrations of 10-20 μg Pb/dL or less (which represents the range of PbB levels of children living near the Site (96)), is not always possible (74).**

LBP remains the most common high-dose source of lead exposure for preschool children even though the use and manufacture of LBP has steadily declined through the 1950s, '60s and '70s. In 1978 the Consumer Product Safety Commission banned the manufacture of paint containing more than 0.06% lead by weight on interior and exterior residential surfaces, toys and furniture. However, LBP is still available for industrial use and may occasionally end up being used for residential purposes (74). Use of LBP (containing up to 50% lead) was widespread during and prior to the 1940s (20, 74) when most of the homes surrounding the Site

were built. This would suggest that LBP may be one of the primary contributors to increased lead concentrations in residential soil, house dust and blood lead in Granite City, which is also supported by the results of the IDPH Study (84).

Site-Specific Case Studies

Although exposure to environmental sources of lead contribute to overall PbB concentrations, the magnitude of this effect varies and may not be responsible for the majority of variation in child PbB levels. This is evident in two of the studies reviewed. An analysis of data collected from New Haven, CT concluded that environmental exposures to lead accounted for only 11.7% of the variation in PbB (20). The study conducted at the NL Industries/TaraCorp. Site, by the Illinois Department of Public Health found that even when all variables were considered only 37% of the variance in PbB was accounted for. Therefore, the majority of variation in blood leads at these sites are unidentified.

Variation in the results, and ultimate conclusions, of studies conducted to assess the potential effects of lead exposure illustrate the intricate nature of evaluating exposure sources and pathways. Therefore, it is important to use these study results to assist in the decision process for establishing the best remedial measure for a specific site. To assist with this decision making process, a three-city study (Baltimore, Boston and Cincinnati) was undertaken in order to assess the effects of lead source abatement following exposure, rather than assessing exposure to contamination.

This study, known as The Urban Soil Lead Abatement Demonstration Project (Three-City Study) was authorized under SARA in 1986 in order to determine whether abatement of lead in soil could reduce the lead in blood of inner city children. Three coordinated longitudinal studies of urban children were conducted to evaluate the capability of intervention into the pathway of lead exposure. This intervention was expected to reduce the children's blood lead concentrations (90).

Individually, the results of these three studies may support three different conclusions concerning the feasibility of reducing lead exposure by abating soil. USEPA has presented a collective review of the results which they feel presents a common picture that places a significant role for

soil abatement in the total scheme of lead exposure. Others in the scientific community do not share this review. USEPA also concluded that the abatement of one source of lead exposure should be considered only in the context of the other sources present. Without attention paid to all sources of lead exposure, the abatement of a single source may help to decrease PbB but this decrease will likely be minimal, temporary and a gross mismanagement of remedial resources (90).

Development of Cleanup Criteria

Although all of the information discussed in this section provides data regarding the potential sources of lead, exposure pathways and the correlation of these exposures to concentrations of lead in blood, it does not present a foundation on which to establish cleanup levels in soil at the Site. The majority of the references introduced into the NL Industries/Taracorp Site Administrative Record do not support

USEPA's proposed cleanup level of 500 ppm. One USEPA guidance document suggests a cleanup range for lead in soil to be set within the range of 500-1000 ppm. However, this guidance also states that a site may deviate from this range based upon site-specific conditions (73).

Several of the references reviewed suggested that lead soil concentrations in excess of the USEPA's guidance for residential soil (i.e. 500-1000 ppm) may contribute an unacceptable level of health risk via direct or indirect effects on blood lead concentrations (20, 71, 72, 74, 104 105, 112). However, USEPA guidance (OSWER Directive # 9355.4-12) for defining a cleanup level within this range requires the use of the IEUBK model for soils with lead concentrations greater than the screening level of 400 ppm. The screening level is not to be viewed as a cleanup goal. Rather, it is to be used as a tool to determine which sites or portions of sites do not require further study and to encourage voluntary cleanup. Screening levels are defined as a level of contamination above which there may be enough concern to warrant a site-specific study of risks. Levels of contamination above the screening level would not automatically require a removal action, nor designate a site as "contaminated" (107, 108).

This USEPA guidance recommends that preliminary remediation goals (PRGs) for lead contaminated soils be developed using the IEUBK model on a site-specific basis, where site data support the modification of model default parameters. For some Superfund sites, the use of site-specific soil and dust characteristics resulted in PRGs that were more than twice the screening level (107).

The guidance adds that it is more important to note that the model alone does not determine the cleanup levels required at a site. Additional considerations must include other factors such as costs of remedial options, reliability of institutional controls, technical feasibility, and/or community acceptance. Once all of these factors are addressed, higher cleanup levels may be selected (107).

It does not appear that USEPA has complied with this guidance in developing the proposed soil cleanup level of 500 ppm. Although the IEUBK model was employed by USEPA in their effort to establish soil cleanup concentrations, the default parameters were not replaced with site-specific data with the exception of a calculated soil to dust transport factor which suggested that 70% of the lead content in house dust is attributed to lead in soil (112). This assumption was not supported by the IDPH study (84).

In conclusion, the documents reviewed do not support the recommendation of a 500 ppm cleanup level for lead in residential soils as a mechanism for eliminating health risks associated with elevated blood lead levels at the NL Industries/Taracorp Site. The information reviewed supports the concept that cleanup levels should be determined for each Site using site-specific data along with available data analysis techniques and current toxicological information to estimate a reasonable cleanup level based upon protecting human-health and the environment. This analysis includes the incorporation of site-specific information into the IEUBK and other computerized statistical modeling approaches. Once the proposed cleanup guidance has been developed, it should then be subject to feasibility evaluation by the scientific, regulatory and public communities which will be affected by its implementation. The consideration of health-related, economic, social and scientific factors must be weighed prior to finalization of any cleanup standard in order to determine the most technically prudent and cost-effective remedial action for the Site.

6.0 BIBLIOGRAPHY

1 Angle, C. R., Kuehneman, A. J., and Manton, W. H. *Kinetics of Childhood Lead: The Omaha Duplicate Diet Study* - Draft Report.

2 Bornschein, R. L., Clark, C. S., Grote, J., Peace, B., Roda, S., and Succop, P. Soil Lead - Blood Lead Relationship in a Former Lead Mining Town, *Lead in Soil: Issues and Guidelines*:149

3 Lutz, P. M., Javachandran, C., Gale, N. L., Hewett, J., Phillips, P. E., Looney, F. M., and Bengsch, H. *Draft: Immunity in Children with Exposure to Environmental Lead: I. Effects on Cell Numbers and Cell-Mediated Immunity.*

4 National Research Council (NRC). (1993). Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations. Washington, D.C. National Academy Press.

5 Thornton, I, Watt, J. M., Davies, D. J. A., Hunt, A., Cotter-Howells, J., and Johnson, D. L. (1981-1992) *Lead Contamination of U.K. Dusts and Soils and Implications for Childhood Exposure: An Overview of the Work of the Environmental Geochemistry Research Group.* Environmental Geochemistry Research Center for Environmental Technology, Imperial College, London SW7.

6 Kehoe, Robert A. (1961). The Harben Lectures, 1960: The Metabolism of Lead in Man in Health and Disease. *Journal R. I. P. H. & H. I.* May:101-121.

7 Rosen, John F., and Trinidad, Emma E. (1974). Significance of Plasma Lead Levels in Normal and Lead-Intoxicated Children. *Environmental Health Perspective* May:139-144.

8 Barry, P. S. I. (1975). A Comparison of Concentrations of Lead in Human Tissues. Reprinted from *British Journal of Industrial Medicine* 32:119-139.

9 Barry, P. S. I. (1975 - corrected Jan. 1976). Complete Set of Data in Support of "A Comparison of Concentrations of Lead in Human Tissues." Published in *British Journal of Industrial Medicine* 32:119-139.

10 Barry, P. S. I., and Mossman, D. B. (1970). Lead Concentrations in Human Tissues. *British Journal of Industrial Medicine* 27:339-351.

11 Damstra, Terri. (1977). Toxicological Properties of Lead. *Environmental Health Perspectives* 19:297-307.

12 Yankel, Anthony J., and von Lindern, H. (1977). The Silver Valley Lead Study: The Relationship Between Childhood Blood Lead Levels and Environmental Exposure. *Journal of the Air Pollution Control Association* August:27(8):763-767.

13 Ziegler, Ekhard E., Edwards, Barbara B., Jensen, Robert L., Mahaffey, Kathyryn R., and Fomon, Samuel J. (1978). Absorption and Retention of Lead by Infants. *Pediat. Res.* 12:29-34.

14 Barltrop, D., and Meek, F. (1979). Effect of Particle Size on Lead Absorption from the Gut. *Archives of Environmental Health* July/August:280-285.

15 Needleman, Herbert L. (1980). Lead Exposure and Human Health: Recent Data on an Ancient Problem. *Technology Review* March/April:39-45.

16 Barry, P. S. I. (1981). Concentrations of Lead in Tissues of Children. *British Journal of Industrial Medicine* 38:61-71.

17 Barry, P. S. I. (1981). Concentrations of Lead in the Tissue of Children. *British Journal of Industrial Medicine* 38:61-71.

18 Needleman, Herbert L. (1981). The Health Effects of Low Level Exposure to Lead. *Ann. Rev. Public Health*, 277-298.

19 Needleman, Herbert L. (1982). The Neurobehavioral Consequences of Low Lead Exposure in Childhood. *Neurobehavioral Toxicology and Teratology* 4:729-732.

20 Stark, Alice D., Quah, Ruth Fitch, Meigs, J. Wister, and DeLouise, Edward. (1982). The Relationship of Environmental Lead to Blood-Lead Levels in Children. *Environmental Research* 27:372-383.

21 Mahaffey, Kathryn R., Annest, Joseph L., Roberts, Jean, and Murphy, Robert S. (1982). National Estimates of Blood Lead Levels: United States, 1976-1980. Reprinted from the *New England Journal of Medicine* Sept. 2:307:573-579.

22 Kneip, T. J., Mallon, R. P. and Harley, N. H. (1983). Biokinetic Modelling for Mammalian Lead Metabolism. *NeuroToxicology* 4(3):189-192.

23 Needleman, H. L. (1983). Lead at Low Dose and the Behavior of Children. *Acta. Psychiat. Scand.* 67 (Suppl. 303):26-37.

24 Ryu, Jacqueline E., Ekhard, R. D., Ziegler, E., Nelson, Steven E., and Femor, Samuel J. (1983). Dietary Intake of Lead and Blood Lead Concentration in Early Infancy. *Am. J. Dis. Child.* September:137:886-891.

25 Angle, Carol R., Marcus, Allan, Cheng, I-Hui, and McIntire, Matilda S. (1984). Omaha Childhood Blood Lead and Environmental Lead: A Linear Total Exposure Model. *Environmental Research* 35:160-170.

26 Brunekreef, Bert. (1984). The Relationship Between Air Lead and Blood Lead In Children: A Critical Review. *The Science of the Total Environment* 38:79-123.

27 Rabinowitz, Michael, and Needleman, Herbert. (1984). Variability of Blood Lead Concentrations During Infancy. *Archives of Environmental Health* March/April:39(2):74-77.

28 Bornschein, R. L., Hammond, P. B., Dietrich, K. N., Succop, P., Krafft, K., Clark, S., Berger, O., Pearson, D., and Que Hee, S. (1985). The Cincinnati Prospective Study of Low-Level Exposure and Its Effects on Child Development: Protocol and Status Report. *Environmental Research* 38:4-18.

29 Bornschein, R. L., Succop, P., Dietrich, K. N., Clark, C. S., Que Hee, S., and Hammond, P. B. (1985). The Influence of Social and Environmental Factors on Dust Lead, Hand Lead, and Blood Lead Levels in Young Children. *Environmental Research* 38:108-118.

30 Marcus, Allan H. (1985). Multicompartment Kinetic Models for Lead. *Environmental Research* 36:441-458.

31 Rabinowitz, Michael, and Needleman, Herbert. (1985). Lead in Milk and Infant Blood: A Dose-Response Model. *Archives of Environmental Health* Sept./Oct.:40(5):283-286.

32 Bornschein, R. L., Succop, P.A., Kraft, K.M., Clark, C.S., Peace, B. and Hammon, P.B. Exterior Surface Dust Lead, Interior House Dust Lead and Childhood Lead Exposure in an Urban Environment, *Trace Substances in Environmental Heald-XX: Proceedings of the University of Missouri's 20th Annual Conference on Trace Substances in Environmental Health*:322

33 Craswell, P. W., Price, J/. Boyle, P. D., Heazlewood, V. J., Baddeley, H., Lloyd, H. M., Thomas, B. J., Thomas, B. W., and Williams, G. M. (1986). Chronic Lead Nephropathy in Queensland: Alternative Methods of Diagnosis. *Aust. NZ J. Med.* 16:11-16.

34 Koh, T. S., and Babidge, P. J. (1986). A Comparison of Blood Lead Levels in Dogs from a Lead-Mining, Lead-Smelting, Urban and Rural Island Environment. *Australian Veterinary Journal* Sept.:63(9):282-285.

35 Rabinowitz, Michael B., Leviton, Alan, and Needleman, Herbert L. (1986). Occurrence of Elevated Protoporphyrin Levels in Relation to Lead Burden in Infants. *Environmental Research* 39:253-257.

36a United States Environmental Protection Agency. USEPA. (1986). Air Quality Criteria for Lead. Office of Research and Development. Volume II of IV. June.

36b United States Environmental Protection Agency. USEPA. (1986). Air Quality Criteria for Lead. Office of Research and Development. Volume III of IV. June.

36c United States Environmental Protection Agency. USEPA. (1986). Air Quality Criteria for Lead. Office of Research and Development. Volume IV of IV. June.

37 Hamir, A. N., and Handson, P.D. (1986). Time Required for Elevated Blood Lead Concentrations to Return to Normal in Dogs. *Australian Veterinary Journal, Vol.* May:63(5):165-166.

38 Bornschein, R. L., Succop, P. A., Krafft, K. M., Clark, C.S., Peace, B., and Hammond, P. B. (1986). *Exterior Surface Dust Lead, Interior House Dust Lead and Childhood Lead Exposure in an Urban Environment.* Presented at the Conference on Trace Metals in Environmental Health, Columbia, Missouri. June.

39 Bellinger, David, Leviton, Alan, Rabinowitz, Michael, Needleman, Herbert, and Waternaux, Christine. (1986). *Correlates of Low-Level Lead Exposure in Urban Children at 2 Years of Age.* PEDIATRICS June:77(6):826-833.

40 Mahaffey, Kathryn R. Biokinetics of Lead during Pregnancy. *Fundamental and Applied Toxicology* 16:15-16 (1991).

41 Needleman, Herbert L. (1987). Introduction: Biomarkers in Neurodevelopmental Toxicology. *Environmental Perspectives* 74:149-152.

42 Schutz, Andrejs, Skerfving, Staffan, Ranstam, Jonas, and Christoffersson, Jan-Ove. (1987). Kinetics of Lead in Blood After the End of Occupational Exposure. *Scand. J. Work Environ. Health* 13:221-231.

43 Brockhaus, A., Collet, W., Doigner, R., Engelke, R., Ewers, U., Freier, I., Jennann, E., Kramer, U., Manojlvic, N., Turfeld, M., and Winneke, G. (1988). Exposure to Lead and Cadmium of Children Living in Different Areas of North-West Germany: Results of Biological Monitoring Studies 1982-1986. *Occupational Environmental Health* 60:211-222.

44 Fergusson, D. M., Fergusson, J. E., Horwood, L. J., and N. G. Kinzett. (1988). A Longitudinal Study of Dentine Lead Levels, Intelligence, School Performance and Behavior: Part II. Dentine Lead and Cognitive Ability. *J. Child Psychol. Psychiat.* 29(6):793-809.

45 McMichael, A.J., Baghurst, P.A., Wigg, N.R., Vimpani, G.V., Robertson, E.F., Roberts, R.J. Port Pirie Cohort Study: Environmental Exposure to Lead and Children's Abilities at the Age of Four Years. *The New England Journal of Medicine*:468 (1988).

46 Nriagu, Jerome O., and Pacyna, Jozef M. (1988). Quantitative Assessment of Worldwide Contamination of Air, Water and Soils by Trace Metals. *Nature* May 12:333:134-139.

47 Rosen, John F. (1988). "The Toxicological Importance of Lead in Bone: The Evolution and Potential Uses of Bone Lead Measurements by X-Ray Fluorescence to Evaluate Treatment Outcomes in Moderately Lead Toxic Children." Biological Monitoring of Toxic Metals. Plenum Publishing Corp.

48 Davies, Brian E., and Wixson, Bobby G. (1988). *Lead in Soil: Issues and Guidelines*. Proceedings of a conference held in the Hotel Europa in Chapel Hill, NC, USA. March 7-9.

49 Wittmers, Lorentz E., and Aufderheide, Arthur C. (1988). Lead in Bone IV. Distribution of Lead in the Human Skeleton. *Archives of Environmental Health* Nov./Dec.:43(6):381-391.

50 Dietrich, K.N., Krafft, K.M., Bier, M., Berger, O., Succop, P.A., and Bornschein, R.L. Neurobehavioural Effects of Foetal Lead Exposure: The First Year of Life. *Neurobehavioural Effects of Foetal Lead Exposure*: 320.

51 Grant, L. D., and Davis, J. M. Effects of Low-Level Lead Exposure on Paediatric Neurobehavioural Development: Current Findings and Future Directions. Lead Exposure and Child Development.

52 Hansen, Ole Norby, Trillingsgaard, Angen, Beese, Inge, Lyngbye, Troels, and Grandjean, Phillipe. (1989). A Neuropsychological Study of Children With Elevated Dentine Lead Level: Assessment of the Effect of Lead in Different Socio-Economic Groups. *Neurotoxicology and Teratology* 11:205-213.

53 Hatzakis, A., Kokkevi, A., Maravelias, C., Katsouyanni, K., Salaminios, F., Kalandidi, A., Koutselinis, A., Stece Deficits in. *Lead Exposure*:211. Psychometric Intelligence Deficits in Lead-Exposed Children.

54 Madhavan, Shantha, Rosenman, Kenneth D., and Shehata, Terry. (1989). Lead in Soil: Recommended Maximum Permissible Levels. *Environmental Research* 49:136-142.

55 Mushak, P. Biological Monitoring of Lead Exposure in Children: Overview of Selected Biokinetic and Toxicological Issues. *Lead Exposure*:129.

56 Mushak, Paul, and Crocetti, Annemarie F. (1989). Report: *Determination of Numbers of Lead-Exposed American Children as a Function of Lead Source: Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning*. Academic Press. February 21.

57 Mushak, Paul, Davis, Michael, Crocetti, Annemarie F., and Grant, Lester D. (1989). Prenatal and Postnatal Effects of Low-Level Lead Exposure: Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning. *Environmental Research* 50:11-36.

58 Rabinowitz, Michael B., Leviton, Alan, and Bellinger, David C. (1989) Blood Lead - Tooth Lead Relationship Among Boston Children. *Bull. Environ. Contam. Toxicol.* 43:485-492.

59 Thomson, G. O. B., Raab, G. M., Hepburn, W. S., Hunter, R., Fulton, M., and Laxen, D. P. H. (1989). Blood-Lead Levels and Chilldren's Behavior-Results from the Edinburgh Lead Study. *J. Child Psychol. Psychiat.* 30(4):515-528.

60 Rosen, John F., Markowitz, Morri E., Bijur, Polly E., Jenks, Sarah T., Wielopolski, Lucian, Kalef-Ezra, John A., and Slatkin, Daniel N. (1989). L-Line X-ray Fluorescence of Cortical Bone Lead Compared with the $CaNa_2EDTA$ Test in Lead-toxic Children: Public Health Implications. *Proc. Natl. Acad. Sci. USA* January:86:685-689.

61 Dietrich, Kim N., Succop, Paul A, Bornschein, Robert L., Krafft, Kathleen M., Berger, Omer, Hammond, Paul B., and Buncher, Ralph. (1990). Lead Exposure and Neurobehavioral Development in Later Infancy. *Environmental Health Perspectives* 89:3-19.

62 Winneke, Gerhard, Brockhaus, Arthur, Ewers, Ulrich, Kramer, Ursula, and Neuf, Manfred. (1990). Results from the European Multicenter Study on Lead Neurotoxicity in Children: Implications for Risk Assessment. *Neurotoxicology and Teratology* 12:553-559.

63 Mushak, P. (1990). *Gastro-Intestinal Absorption of Lead in Children and Adults: Overview of Biological and Biophysico-Chemical Aspects.* Symposium on the Bioavailability and Dietary Exposure of Lead. Environmental Criteria and Assessment Office, RTP. September 24-27.

64 Marcus, Allan H., and Bernholc, Alissa. (1990). *Inter-Site Comparisons of Environmental Lead Uptake.* Symposium on Bioavailability, USEPA and the Society for Environmental Geochemistry and Health. September.

65 Bellinger, David, Leviton, Alan, Rabinowitz, Michael, Allred, Elizabeth, Needleman, Herbert, and Schoebaum, Stephen. (1991). Weight Gain and Maturity in Fetuses Exposed to Low Levels of Lead. *Environmental Research* 54:151-158.

66 United States Department of Health and Human Services. (1991). Important Facts about Childhood Lead Poisoning Prevention: The 1991 CDC Statement Preventing Lead Poisoning in Young Children. Agency for Toxic Substances and Disease Registry.

67 Mushak, Paul. (1991). Gastro-Intestinal Absorption of Lead in Children and Adults: Overview of Biological and Biophysico-Chemical Aspects. *Chemical Speciation and Bioavailability* 3(314):87-104.

68 Nilsson, U., Attewell, R., Christoffersson, J.-O., Schultz, A., Ahlgren, L., Skerfving, S., and Mattsson. (1991). Kinetics of Lead in Bone and Blood After End of Occupational Exposure. *Pharmacology & Toxicology* 69:477-484.

69 Rabinowitz, Michael B. (1991). Toxicokinetics of Bone Lead. *Environmental Health Perspectives* 91:33-37.

70 Rosen, John F., Markowitz, Morri E., Bijur, Polly E., Jenks, Sarah T., Wielopolski, Lucian, Kalef-Ezra, John A., and Slatkin, Daniel N. (1991). Sequential Measurements of Bone Lead Content by L X-Ray Fluorescence in $CaNa_2EDTA$-Treated Lead-Toxic Children. *Environmental Health Perspective* 91:57-62.

71 United States Environmental Protection Agency. USEPA. (1991). *Testimony of William K. Reilly, Admin., USEPA Before the Subcommittee on Toxic Substances.* Environmental Oversight, Research and Development of the Committee on Environmental and Public Works, US Senate. February 21.

72 United States Environmental Protection Agency. USEPA. (1991). Memorandum re: Final Agency Lead Strategy. Washington, D.C. February 26.

73 United States Environmental Protection Agency. USEPA. (1991). Memorandum re: Update On OSWER Soil Land Cleanup Guidance. Washington, D.C. August 29.

74 Centers for Disease Control and Prevention/ United States Department of Health and Human Services. (1991). *Preventing Lead Poisoning in Young Children.* October.

75 Marcus, Allan. USEPA. (1991). *DRAFT REPORT: Analysis of Lead in Soil and Dust Data*. Office of Toxic Substances, Exposure Evaluation Division. December 3.

76 Pediatrics. Lead Poisoning: From Screening to Primary Prevention. Volume 92, No.1, (July, 1993).

77 Baghurst, Peter A., McMichael, Anthony J., Wigg, Neil R., Vimpani, Graham V., Robertson, Evelyn F., Roberts, Russell J., and Tong, Shi-Lu. (1992). Environmental Exposure to Lead and Children's Intelligence at the Age of Seven Years. *The New England Journal of Medicine* October 29:327(18):1279-1284.

78 Beck, Barbara D. (1992). Symposium Overview: An Update on Exposure and Effects of Lead. *Fundamental and Applied Toxicology* 18:1-16.

79 Bellinger, David C., Stiles, Karen M., and Needleman, Herbert L. (1992). Low-Level Lead Exposure, Intelligence and Academic Achievement: A Long-term Follow-up Study. *PEDIATRICS* December:90(6):855-861.

80 Freeman, G. B., Johnson, J. D., Killinger, J. M., Liao, S. C., Feder, P. I., Davis, A. O., Ruby, M. V., Chaney, R. L., Lovre, S. C., and Bergstrom, P. D. (1992). Relative Bioavailabilty of Lead from Mining Waste Soil in Rats. *Fundamental and Applied Toxicology* 19:001-0011.

81 Rothenberg, Perez-Guerrero, Schnaas-Arrieta, Perroni-Hernandez, Torres-Mercado, Gomez-Ruiz Consuelo, and Martinez-Medina. (1992). *Simple Modeling of Maternal Lead Levels During Pregnancy: The Role of Extrinsic and Intrinsic Factors*. Poster presented at Interntional Conference on Lead & Other Trace Substances. Society for Environmental Geochemistry & Health, Research Triangle Pk, NC. August 3.

82 Marcus, Allan H. (1992). *Comparative Approaches to Superfund Site Assessments for Young Children Exposed to Lead (transparencies from overhead presentation).* Society for Environmental Geochemistry and Health. August 3-4.

83 Wasserman, G., Graziano, J. H., Factor-Litvak, P., Popovac, D., Morina, N., Musabegovic, A., Vrenezi, No., Capuni-Paracka, S., Lekic, V., Preteni-Redjepi, E., Hadzialijevic, S., Slavkovich, V., Kline, J., Shrout, P., and Stein, Z. (1992). Independent Effects of Lead Exposure and Iron Deficiency Anemia on Developmental Outcome at Age 2 Years. *The Journal of Pediatrics* November:121(5):Part I:695-703.

84 Illinois Dept. of Public Health. (1992). Draft Report: *Madison County Granite City, IL Lead Exposure Study*. U S Dept. of Health and Human Services, Atlanta, GA. December 8.

85 Dietrich, Kim N., Berger, Omer G., Succop, Paul A., Hammond, Paul B., and Bornschein, Robert L. (1993). The Developmental Consequences of Low to Moderate Prenatal and Postnatal Lead Exposure: Intellectual Attainment in the Cincinnati Lead Study Cohort Following School Entry. *Neurotoxicology and Teratology* 15:37-44.

86 United States Department of Health and Human Services. (1993). Toxicological Profile for Lead.

87 Ruff, Holly A., Bijur, Polly E., Markowitz, Morri, Ma, Yeou Cheng, and Rosen, John F. (1993). Declining Blood Lead Levels and Cognitive Changes in Moderately Lead-Poisoned Children. *JAMA* April 7:260(13):1541-1646.

88 Long, Thomas IDPH. (1993). Letter to F. Stallings, ATSDR, Re: Request for ATSDR's Review of the Multi-State Lead Exposure Study. Illinois Department of Public Health. April 14.

89 Frank, A. (1993). Peer Review Comment Forms to ATSDR Re: (1) Lead and Cadmium Exposure Study; (2) Lead Exposure Study, Madison County; and (3) Management of Children with Slightly Elevated Blood Lead Levels. Agency for Toxic Substance and Disease Registry Atlanta, GA. June 8.

90 United States Environmental Protection Agency. USEPA. (1993). *Review Draft: Urban Soil Lead Abatement Demonstration Project: Volume I: Ingegrated Report.* Office of Research and Development, Washington, D.C. EPA/600/AP-93/001a. July.

91 United States Environmental Protection Agency. USEPA. (1993). *Urban Soil Lead Abatement Demonstration Project: Volume II: Boston Report.* Office of Health and Environmental Assessment, Washington, D.C. EPA/600/AP-93/001B. July.

92 United States Environmental Protection Agency. USEPA. (1993). *Urban Soil Lead Abatement Demonstration Project: Volume III: Baltimore Report.* Office of Health and Environmental Assessment, Washington, D.C. EPA/600/AP-93/001C. July.

93 United States Environmental Protection Agency. USEPA. (1993). *Urban Soil Lead Abatement Demonstration Project: Volume IV: Cincinnati Report.* Office of Health and Environmental Assessment, Washington, D.C. EPA/600/AP-93/001D. July.

394 Londergan, Richard J. (1993). Peer Review Comment Forms to ATSDR Re: (1) Lead and Cadmium Exposure Study; (2) Lead Exposure Study, Madison County; and (3) Management of Children with Slightly Elevated Blood Lead Levels. U S Department of Health and Human Services. Agency for Toxic Substance and Disease Registry, Atlanta, GA. July 23.

95 Marcus, Allan, and Elias, Robert W. Estimating the Contribution of Lead-Based Paint to Soil Lead, Dust Lead, and Childhood Blood Lead.

96 United States Department of Health and Human Services. (1994). *Madison County Lead Exposure Study, Granite City, IL.* Illinois Department of Public Health, Springfield, IL. February.

97 United States Environmental Protection Agency/The Technical Review Workgroup for Lead. USEPA. (1994). *Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children.* Office of Emergency and Remedial Response, Washington, D.C. EPA-540/R-93/081. February 1994.

98 Adler, Jonathan H., Needleman, Herbert L., and Miller, Jeffrey T. (1994). Editorial: Getting the Lead Out. *Garbage* Spring:7-8.

99 Stipp, David. (1994). Probe Finds Errors, but No Misconduct, In Work by Lead-Poisoning Researcher. *Wall Street Journal* March 9.

100 Longest II, Henry L., and Shapiro, Michael H. USEPA. (1994). Memorandum Re: Transmittal of Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children and IEUBK Model, Version 0.99d. March 23.

101 Rabinowitz, Michael, and Needleman, Herbert. (1984). Variability of Blood Lead Concentrations During Infancy. *Archives of Environmental Health* March/April:39(2):74-77.

102 Marcus, Allan H., Hogan, Karen, White, Paul, and Van Leeuwen, Patricia. USEPA. (1994). Comments on Madison County Lead Exposure Study, Granite City, IL. May 23.

103 Van Leeuwen, Patricia. USEPA. (1994). Cover letter to John R. Lumpkin, IDPH, Re: USEPA Comments on the Draft Lead Exposure Study. May 24.

104 United States Department of Health and Human Services. (1994). *Draft: Multisite Lead and Cadmium Exposure Study with Biological Markers Incorporated.* Public Health Service, Agency for Toxic Substances and Disease Registry, Atlanta, GA. June.

105 von Lindern, Ian H. (1994). *Reducing Children's Blood Lead Levels at the Bunker Hill Superfund Site in Northern Idaho, USA Through Health Intervention and Soil/Dust Source Control Measures.* Presented at the 1994 International Lead Abatement and Remediation Conference, Newcastle, New South Wales, Australia. June 3.

106 Siegel, Steven. USEPA. (1994). Cover letter to Dave Webb, IDPH, Re: Draft Lead Exposure Study. June 15.

107 United States Environmental Protection Agency. USEPA. (1994). Memorandum Re: Revised Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities. Office of Solid Waste and Emergency Response, Washington, D.C. July 14.

108 United States Environmental Protection Agency. USEPA. (1994). Memorandum Re: Guidance on Residential Lead-Based Paint, Lead-Contaminated Dust, and Lead-Contaminated Soil. Office of Prevention, Pesticides and Toxic Substances, Washington, D.C. July 14, 1994.

109 Brody, Debra J., Pirkle, James L., Kramer, Rachel A., Flegal, Katherine M., Matte, Thomas D., Gunter, Elaine W., and Paschal, Daniel C. (1994). Blood Lead Levels in the US Population. *JAMA* July 27:272:4:277-283.

110 Goldman, Lynn R., and Carra, Joseph. (1994). Editorial: Childhood Lead Poisoning in 1994. *JAMA* July 27:272(4):315-316.

111 Pirkle, James L., Brody, Debra J., Gunter, Elaine W., Kramer, Rachel A., Paschal, Daniel C., Flegal, Katherine M., and Matte, Thomas D. (1994). The Decline in Blood Lead Levels in the United States: The National Health and Nutrition Examination Surveys (NHANES). *JAMA* July 27:272(4):284-291.

112 Marcus, Allan H. *Preliminary Assessment of Data From the Madison County Lead Study and Implications for Remediation of Lead-Contaminated Soil.* Environmental Criteria and Assessment Office.

113 Chemical Speciation and Bioavailability. (1991). *Proceedings of the Symposium on the Bioavailability and Dietary Exposure of Lead, September 1990, Chapel Hill, NC* December:3(3/4).

114 Croceti, Annemarie F., Mushak, Paul, and Schwartz, Joel. (1990). Determination of Numbers of Lead-Exposed Women of Childbearing Age and Pregnant Women: An Integrated Summary of a Report to the U.S. Congress on Childhood Lead Poisoning. *Environmental Health Perspectives* 89:121-124.

115 United States Environmental Protection Agency. USEPA. (1994). Proposed Plan: NL Industries/Taracorp Superfund Site, Granite City, Illinois. Office of Public Affairs, Region 5, Chicago, IL. October 11, 1994.

EXHIBIT B

REVIEW OF THE MADISON COUNTY LEAD EXPOSURE STUDY AND RELATED DOCUMENTS

January 12, 1995

The ChemRisk® Division of McLaren/Hart Environmental Engineering Corporation (McLaren/Hart) reviewed the February 1994 Madison County Lead Exposure Study prepared by the Illinois Department of Public Health (Exposure Study), the May 1994 "Comments" by Allan Marcus et al. (USEPA Comments), the July 1994 memo from LeVois to Long responding to the USEPA Comments (the LeVois Response), and the October 1994 "Preliminary Assessment of Data from the Madison County Lead Study and Implications for Remediation of Lead-Contaminated Soil" by Allan Marcus (the Marcus Reassessment). This review centered on identifying the main issues for discussion, commenting on study design and analysis issues in general and with specific reference to particular documents, and providing some recommendations for additional analyses that could be performed in the future to more clearly answer what are believed to be the relevant questions.

Conclusions of the McLaren/Hart Review

A number of conclusions can be drawn regarding McLaren/Hart's review. The most noteworthy are:

- The study was considered of high quality and all conclusions reached in the Exposure Study were supported by the data and the statistical analyses performed.
- Strong rebuttals were made to the majority of criticisms made in the USEPA Comments.
- McLaren/Hart answered the three key questions posed in the beginning of that report as follows:

  "Based on the analyses performed in the original study and barring changes in interpretation due to additional treatment of the data:"

  - The lead levels in children's blood in the Madison County study area do <u>not</u> indicate an imminent public health problem.
  - Soil remediation is <u>not</u> likely to significantly reduce blood lead levels in children, in general.

- Soil remediation is not likely to significantly reduce blood lead levels in children with "elevated" levels of blood lead.

Main Issues

McLaren/Hart believes that the Exposure Study and commentaries should have focussed on answering three main questions relevant to the public health issues surrounding cleanup in the vicinity of the NL Industries/TaraCorp Superfund Site:

(1) Do lead levels in children's blood indicate an imminent public health problem in the Madison County study area?

(2) Is soil remediation likely to result in reductions in children's blood lead levels in general?

(3) Is soil remediation likely to result in reductions in blood lead levels among those children with "elevated" levels (i.e., greater than 10 μg/dl)?

The key to answering these questions is data interpretation. Because of the complexity of the relationships among biological measures, environmental measures, behavioral factors, and socioeconomic factors, there is heavy reliance on specialized statistical techniques to interpret data collected in the Exposure Study. As evidenced by the Exposure Study, USEPA comments, the LeVois Response, and the Marcus Reassessment, choice of appropriate statistical techniques and scrutiny of their results are not straightforward matters. In some cases, however, the exchange of expert opinion on which analyses approach to use failed to center on the issues of primary importance in evaluating soil cleanup options.

Exposure Study Design Issues

It should be noted that a study requiring voluntary consent and participation is much more difficult to conduct than a controlled laboratory study involving a random sample from some population. During this review there were no serious unavoidable problems in recruitment or participation found within the Exposure Study. The researchers have done as well as possible given the many limitations encountered with studies of this type. The Exposure Study's data analysis is sensitive to study design limitations. However, when interpreting and attempting to generalize the study results, it is important to remember that the study participants do not represent a random sample from the community.

It is a clearly a matter of disagreement whether the nonsampled proportion of the population would likely have similar, higher, or lower blood lead levels than the study participants. In the Exposure Study it is argued that recruitment was concentrated in areas where highest smelter-related exposures were expected. The USEPA Comments suggest that exposures and/or blood lead levels are likely higher among the nonsampled population than among the studied population. It is impossible to resolve this question without understanding the characteristics of the nonparticipants within the study area. Therefore, if generalizing to the community is important, then some demonstration is required that the participants are similar to or different from the nonparticipants as defined by variables in the study (e.g., housing condition, demographics, soil lead levels, etc).

Evaluation of Regression Analyses in the Exposure Study

In general, the presentation of regression results in the Exposure Study is somewhat incomplete, and there are some analyses not presented that could be informative. First of all, more emphasis on evaluating the hierarchical regressions as opposed to the stepwise regressions is suggested. The latter are simply the result of numerical optimizations; the former are designed using scientific beliefs about the process at hand.

The point demonstrated by Table 11 in the Exposure Study is that the addition of soil concentration to the predictive model for log blood lead in children once paint lead and house condition are included allows explanation of only 3 percent more variance in log blood lead. While this is important information, certainly additional questions could be posed and answered, such as what percent of variance is explained upon adding lead in paint and condition of residence when soil composition is in the model? A statement in the Exposure Study abstract says that 11 percent of variance in blood lead is accounted for by lead in paint and condition of houses together; this may be the answer to this question but this is not clear.

Inclusion of some regression diagnostics would be very helpful for those attempting to interpret the analyses. From the information provided, there is no way to verify that the regressions were well-behaved and satisfied the required assumptions. This could also be readily addressed in a revision of the study results.

Within the various documents, there is a debate regarding the importance of age in the analyses. It appears that age is a proxy for exposure and is not pathway-specific (i.e., not specific to paint or soil). Therefore, it should be included somehow in the modeling, either as a continuous or categorical predictor, and using a nonlinear form as necessary because behavioral information would suggest a non-monotone relationship between exposure potential and numerical age.

The Exposure Study does not include terms for interactions among variables within the regression models. Some potential informative interactions might include those between soil and paint lead levels and also between age and soil and/or age and paint to account for some of the age-dependent exposure that is expected.

The USEPA Comments and the Marcus Reassessment concentrate a great deal on distance from the Site smelter as a predictor. Distance from the smelter as measured in the Exposure Study and as used by USEPA is a crude predictor. A better representation would take lead fate and transport issues into account rather than simply raw distance as estimated from a map. Distance in some form may be a proxy for indirect exposure to the Site lead, but as pointed out in the LeVois Response to USEPA's Comments, use of distance in the modeling is a minor consideration when actual soil data is available. Also, it must be recognized that distance, even if considered in an ideal fate and transport sense, could not be a complete exposure proxy because children do not reside exclusively at any particular distance from the Site.

Is there a public health problem?

It would appear that determining whether there is a public health problem with blood lead in children depends on the population of interest and the definition of "problem." Focusing on the studied population, it would appear that there is not a problem with average or typical blood lead levels. Means (arithmetic and geometric) for blood lead levels are at an acceptable level (i.e., less than 10 μg/dl) within the studied population, even among those children whose soil lead exceed the 500 ppm proposed cleanup level. Because the study population is not a random sample from the community in general, extending these results beyond the studied population

is questionable; however, if one accepts that the population fraction not studied is essentially similar to the studied population, then one can infer that average or typical blood lead levels do not indicate a significant risk at the community-wide level.

USEPA raises the concern in their comments that more than 5% of children have blood lead levels exceeding 10 μg/dl. This is an issue regarding the extreme values or tails of the distribution. There would be two main ways to mathematically achieve a distribution with only 5 percent of the values exceeding 10 μg/dl: reducing the mean while keeping the variance constant (i.e., shifting the distribution to lower values while retaining essentially the same shape), or reducing the variance while keeping the mean constant (i.e., tightening the distribution around the existing mean). We know from the low R^2 in the various regression models that behavioral and/or completely unmeasured (and potentially unmeasurable) factors--not environmental lead levels either in paint, soil, or water--account for the overwhelming fraction of variance in children's blood lead levels. Recognizing thus that soil remediation is unlikely to substantially affect the variance in blood lead levels, in order to meet the USEPA target, the entire distribution of blood lead levels would need to be shifted towards lower values. There is likely little clinical public health benefit in lowering the mean blood lead level among Madison County children. A better use of resources would be to target and identify those factors which result in blood lead levels near or exceeding clinically significant values. Neither the Exposure Study nor the Marcus Reassessment demonstrates conclusively that soil remediation could result in lower blood lead levels among children with levels exceeding 10 μg/dl.

Another important consideration about meeting the USEPA target is whether the assumed lognormal model for blood lead concentrations is appropriate. The range that variables can take within the lognormal model is from 0 to infinity. While 0 is a plausible lower bound, an unconstrained upper bound is certainly not plausible from a biological perspective. As the amount of data increases, the fact that biologically implausible extreme values do not occur becomes evident. The failure of the heavy-tailed lognormal model to provide a good fit to the tails of the "real" data can thus become a problem. This may be an important consideration in light of Figure 18 in the Marcus Reassessment which presents cumulative distributions of predicted and observed log blood lead concentrations. Note that while the predicted and observed values tend to align well for the central 60 percent of the data, the upper and lower

20 percent are fit much less well. Concentrating on the upper tail, the portion of the data of most concern to USEPA, note that the predicted values for the upper 20 percent of the data all exceed the observed values, suggesting that the lognormal model may indeed be too heavy-tailed for these data. At the 95th percentile, blood lead levels are overpredicted by the model by about 4 μg/dl or 25 percent; at the maximum, the overprediction is about 70 μg/dl, more than 100 percent of the observed maximum! The lighter-tailed and constrained Weibul model may provide a better fit to these data than the lognormal which clearly is poor in fitting the tails. Alternatively, since it is the extreme values which are of concern, perhaps the modeling should be restricted to the upper tail values using established statistical extreme value models.

In the Marcus Reassessment, Section 3.2, it is stated that assuming a 70 percent soil-to-dust coefficient in the IEUBK lead model results in a very good fit to the Madison County blood lead data. Two comments can be made regarding this statement. First, "very good fit" is a subjective judgment. If the fits were perfect, then the points within Figures 19 and 20 would all fall on a line with unit slope. That is, predicted values would exactly equal observed values in each case. Instead, in Figures 19 and 20 we see a cloud of points with perhaps a visually perceptible positive slope, but certainly not a strong relationship along the unit slope line sketched into each plot. Second, even if there were a "very good fit," this fact alone would not strongly support the 70 percent soil-to-dust coefficient being correct in the IEUBK model. The value of this coefficient could merely be compensating for some other poorly understood, non-modeled, or misspecified parameter within the model. That 70 percent gives the best fit under the circumstances does not mean that 70 percent is the true value.

Recommendations for Additional Analyses

A wealth of data was collected in the Exposure Study and analyzed effectively for the purpose of characterizing lead levels in Madison County children and understanding environmental contributions to lead exposure. For answering the critical questions with respect to a soil remediation decision, there remain several types of analyses that could exploit the existing data. A few of these are suggested below:

We endorse the suggestion in the USEPA Comments to examine the data using structural equation modeling (i.e., path analysis). This is not viewed as a necessity, but should be considered if additional analyses are planned. This approach is a method of evaluating a network of dependent relationships and thus would be quite appropriate for handling the intermeshed predictors of blood lead concentration measured in the Exposure Study. In this case, we have exposure pathway models that could be built and tested; the diagram in the USEPA Comments is one potential structure. Structural equation modeling could check sequential, model-based significance of many of the measured variables. Advantages of the technique include its ability to disconfirm hypothesized models and the ability to employ so-called latent variables that condense information and increase model generality. Potential problems with applying structural equation modeling in this setting include that the models are complex and data-intensive. While complexity may be required to understand truly complex situations, in the event that a plausible model can be developed it does not implicitly affirm causal relationships or overall validity or reliability of the modeling approach.

It is recommended that any additional analysis should (a) include/allow/evaluate statistical interactions among variables; (b) control/include age as a proxy for exposure; (c) use fate and transport modeling, not just distance, to represent indirect exposure to lead from the Site.

There may be some value in analyzing separately the data from children with blood lead levels greater than 10 μg/dl to evaluate how important lead in soil and paint are to their blood lead levels. If statistical modeling cannot demonstrate a strong significant relationship between soil lead levels and blood lead levels among this group that might benefit most from reduced blood lead levels, then there is little chance that remediating soil will produce a health-benefitting reduction of blood lead levels in any segment of the Madison County child population. To evaluate sensitivity of the coefficients for predictor variables developed in this modeling, membership of the group could be expanded in 1 μg/dl increments downward.

Finally, a statistical test of whether blood lead levels differ as a function of age could be enlightening, both as part of the above-suggested analysis and using the full dataset.

Conclusions

(1) Even though additional statistical analyses have been suggested for consideration throughout this review, the Exposure Study is considered to be of high quality.

(2) All nine conclusions reached in the Exposure Study (page 53 and 54 of the Exposure Study) are supported by the existing data and data analyses performed.

(3) A number of the comments made and analyses conducted by the USEPA are not appropriate.

(4) Based on the analyses performed in the Exposure Study and barring changes in interpretation due to additional treatment of the data, the three main questions identified at the beginning of this report can be answered as follows:

- The lead levels in children's blood in the Madison County study area do not indicate an imminent public health problem.
- Soil remediation is not likely to significantly reduce blood lead levels in children, in general.
- Soil remediation is not likely to significantly reduce blood lead levels in children with "elevated" levels of blood lead.

January 13, 1995

NL Industries/Taracorp Site, Granite City, Illinois: Comments Addressing The USEPA's Use Of The IEUBK Model To Justify 500 PPM/Pb Soil Clean-Up Level

By:

Morgan, Lewis & Bockius
2000 One Logan Square
Philadelphia, PA 19103-6993

BACKGROUND

In March of 1990 the United States Environmental Protection Agency ("EPA") issued a record of decision for the NL Industries/TaraCorp site in Madison County, Illinois requiring a residential soil clean up level for lead of 500 ppm. In August and September of 1991, the Illinois Department of Public Health conducted a survey of blood lead, soil lead and lead in indoor dust, as well as other pertinent behavioral and sociological factors, for a sample of children under the age of 6 living at various distances from the site, a draft of which was released in February of 1994 (the "Study"). In May of 1994, EPA reviewers, headed by Allan H. Marcus, issued a report entitled "Comments on Madison County Lead Exposure Study, Granite City, Illinois" (the "Comments"), criticizing many aspects of the Study. Subsequently the EPA, through Marcus, issued a document entitled "Preliminary Assessment of Data from the Madison County Lead Study and Implications for Remediation of Lead Contaminated Soil" (the "Preliminary Assessment"). Without supporting technical details, this document asserted that the EPA Integrated Exposure Uptake

Biokenetic Model for Lead ("IEUBK Model"), as applied in light of the site specific information gathered in the Study, supported the proposed soil remediation level of 500 ppm.

The following comments point out the major flaws in the EPA's assessment of the Site and use of the IEUBK Model to justify its 500 ppm clean up level.

GENERAL CONCLUSIONS

The critical and major point in this critique is that the Study does not demonstrate that the NL/TaraCorp site contributes today to elevated blood lead levels or soil lead levels in the subjects tested, because these levels are no higher than would be expected for a comparable, comparison urban neighborhood. Good science dictates that the agency first identify a reasonable "control" or background level in order to understand whether there are in fact "elevated" levels detected by the surveys. Moreover, the EPA has not demonstrated that a reduction of the soil lead in this area to 500 ppm would result in a significant reduction of childhood blood leads. Indeed, the EPA's course is contrary to the conclusions of the Study, which states that "[e]liminating a variable such as soil that accounted for only 3% of the variance [in blood leads] may only result in a minimal change in measured blood lead levels without any clinical significance." Study at 49.

SPECIFIC COMMENTS

1. **HAS IT BEEN DEMONSTRATED THAT THE SITE CAUSED CURRENT LEAD LEVELS?**

There appears to be some confusion over the EPA's goal in setting a 500 ppm soil remediation level at this site. Is the agency trying to justify this clean up because lead from the site contributed to elevations in blood and soil lead in this neighborhood today, or is EPA simply justifying the clean up because these levels are elevated over the EPA's "ideal," regardless of source? If the latter is the goal, then how can the EPA justify requiring the private parties associated with the site to fund the clean up?

The EPA's stated "ideal" is to have soil lead below 500 ppm and blood leads of 95% of children under 6 years of age below 10 μg/dl. It is highly unlikely that any industrial, urban area with older housing would meet that ideal today. Urban areas generally have higher lead levels because of their history of heavy truck and automobile traffic (lead gasoline residue), older housing (lead paint) and various industries. The EPA might make a policy determination to clean urban soils to 500 ppm, but considering the numerous potential sources of lead in the urban environment, they should be required to prove the source before ordering private parties to fund the clean up. For example, the Study found that for houses with soil leads above 500 ppm the geometric mean outdoor paint lead level was 8.6, but for houses with soil leads less than 500 the mean outdoor paint lead was

3.0. This suggests that outdoor lead paint contributes at least in part to elevated soil readings.

2. CONSIDERATION OF AND THE FAILURE TO IDENTIFY APPROPRIATE BACKGROUND LEVELS

The Study indicates that it was unable to find an appropriate "control" population similar to the Study population in all respects except for the presence of the site. Thus, none of the documents reviewed use actual soil, dust and blood lead data from a control group. There is no meaningful background against which to compare the soil leads and the blood leads to determine if the site had any effect above background. It seems as if a comparable urban area should exist from which actual samples could be taken, perhaps in a nearby city.

In the absence of true background testing, the EPA has not proved that lead levels in this neighborhood are elevated over expected background. The blood leads in the Study were in keeping with national averages for urban children. In determining whether the presence of the site has caused elevated blood lead levels in children in Granite City, the EPA unrealistically compares the data from the Study to the EPA's ideal situation - i.e. 95% of children under 6 with blood leads less that 10 μg/dl. In the Study as a whole 16% of the 490

children tested had blood leads over 10 μg/dl.[1/] The data from the Study are quite consistent with blood lead levels found nationally in children in urban areas as reported in the NHANES III dataset, in which 16.4% of children had blood leads exceeding 10 μg/dl in urban areas of less than one million. Since the percentages are virtually identical, there is no evidence that the presence of the site has had the effect of elevating childhood blood leads in Granite City today, as EPA claims.

Likewise, soil levels do not appear to be much higher than would be expected in a comparable urban area. In the Study 375 soil samples were analyzed, with a mean of 450 ppm and a range from 37 to 3,010 ppm. Study at 33. The Study states that this is "above background levels," comparing to asserted "national" average soil levels of less than 1 to 200 ppm. The use of the "national" average hardly seems a realistic or fair comparison background level for an urban area. A "national" average is generally recognized as being skewed downward by the inclusion of rural and suburban areas. In the absence of actual background data, these soil levels must be compared to similar urban areas with like amounts of lead paint, traffic patterns and other industry. For an urban area such as Philadelphia, for

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1/ The EPA claims in its Comments that the "downwind" area with the highest potential deposition from the smelter had 26% of children over 10 μg/dl (10 out of 39). However, this analysis is not done in the Study and there is no data to support selection of the downwind area. All other areas have less than 16% of the children over 10. However, it is certainly not indicated that the EPA seeks to limit its soil remediation only to the downwind area.

example, a mean of 450 ppm would be below the "background" level and cleaner than many sections of the City where there is aging housing stock with deteriorating lead paint inside and outside, a history of traffic and the effects of a variety of other contributing sources, such as incinerators, sandblasting, etc.

3. SOCIOECONOMIC STATUS AND CONDITION OF HOUSING

The EPA does not account for the effect on blood lead levels of socioeconomic status and condition of housing, but at least by implication concludes that elevated blood leads are due to soil. However, the Study found that "[a]s distance from the smelter increased, the conditions of the houses improved, fewer houses had peeling paint and most houses were owned rather than rented. Furthermore, the education level of the parents increased, the number of smokers and the amount of smoking decreased, the use of air-conditioning increased and other behavioral variables also changed with distance." Study at 45. The Study also found that "[b]uilding condition was one of the better predictors of blood lead in this population." Study at 39. Thus, children living in residences in "good" condition had average blood leads of 6 μg/dl, while children living in residences in poor condition had a mean blood lead level of 11.8.

The EPA's own documents recognize that socioeconomic status and housing condition can play a large part in childhood blood lead levels. "The blood lead levels of two children with

identical lead exposure scenarios, but living in different family behavior patterns might vary greatly. The difference in socioeconomic status might be reflected in differences in household repair and cleaning, washing of children's hands and toys, food preparation methods, concern for balanced meals and improved nutritional status, more regular eating patterns, etc., all of which may impact blood lead levels." Guidance Manual § 4.5.3.2. Therefore, if these factors have a significant influence on blood lead, then they might account for any appearance of higher blood leads closer to the smelter. Likewise, if these are the true causes of any elevations in blood lead, then remediation of soils alone is unlikely to have a significant effect in reducing blood leads.

4. DEVOTING RESOURCES TO EDUCATION

The Study followed up on those children found to have blood leads over 10 μg/dl and provided counselling parents on pathways of lead exposure. These children were retested periodically and showed "a marked and persistent decline in blood lead levels." Study at 51. The EPA criticized this follow-up on the ground that it was not a scientific testing of the hypothesis that education and counselling can reduce blood levels. Comments § 1.3. Rather than dismissing the potential effectiveness of this approach, the EPA should investigate this alternative, which has the benefit of working regardless of the source of the lead

(i.e. soil or lead paint), being less expensive and being less disruptive of the neighborhood. Counselling and education have been used successfully at the Bunker Hill site, for example.

5. USE OF THE IEUBK MODEL IS CONTRARY TO EPA GUIDANCE

The EPA's use of the IEUBK model to set a 500 ppm clean-up level for residential soils is contrary to the EPA's own guidance on the use of the model. The EPA's Guidance Manual states that use of the model to assess trigger levels for soil abatement at the community, regional or state level "is discouraged, because risks cannot be estimated adequately." Guidance Manual at § 4.5.2.4.

6. THE MODEL IGNORES LEAD PAINT

In the public hearing on October 25 and 26, 1994 EPA's representatives admitted that they did not include lead paint in their application of the model. Transcript 10/26/94 at 27, 30 ("if we were to add paint in, the children should be given greater amounts of the lead.") **The EPA's Guidance Manual cautions against indiscriminant use of the model in areas with lead paint.** "If household lead-based paint contribution is highly variable in a community, care should be taken to avoid combining all homes in a single run of the IEUBK Model, as the output results may not be applicable to the population. . . . Children can eat chips or strips of deteriorating lead-based

paint directly from painted surfaces, even when the total area of lead-painted surfaces is so small that the total contribution of lead-based paint to interior household dust or exterior soil is to small to identify." Guidance Manual § 4.5.3.2.

The EPA cannot justify its position that its model, which attributes lead intake to soil, compares favorably to the real data from the Study because the Study participants were exposed to lead paint which the EPA did not account for. It is known that the area studied contains a significant amount of lead paint and at least some of the housing is in poor condition. The Study notes some extremely high and irregularly distributed dust levels, including a maximum value of 71,000 ppm. This suggests a lead paint contribution to some house dust. Therefore, a substantial portion of the measured blood leads must be caused by lead paint.[2/] The EPA's model ignores the contribution of lead paint, thereby overpredicting the magnitude of the contribution from soil. Consequently, lowering the lead in the soil will not lower the blood leads to the same degree asserted by the EPA.

2/ The comments by the Chemrisk division of McLaren Hart point out that the model overpredicts blood leads to a greater degree as it moves toward the upper percent of the data, so that at the 95th percentile blood lead levels are overpredicted by 4 μg/dl. Chemrisk at 6. Since the EPA's goal is to bring the upper percentile children down under 10, if it is overpredicting the blood leads of the upper percentile children, then it is also overpredicting the amount by which soil lead levels would have to be decreased in order to bring these upper percentile children under 10.

7. SOIL LEAD TO DUST LEAD CONCENTRATIONS

The EPA's Preliminary Assessment section 3.2 states that it assumes a 70% soil to dust coefficient, which is understood to mean that 70% of the lead in outdoor soil would be transported into indoor dust. The lead that is taken up by children under the model comes largely from house dust. Thus, if the EPA is wrong about the proportion of lead in soil that goes into house dust, then reduction of the soil lead will not have as great an effect on blood leads as predicted by the model. The EPA's Science Advisory Board Review of the IEUBK Model dated March of 1992 ("SAB Report") emphasized the that different communities may have higher or lower uptakes from the identical levels of soil lead and that an important factor explaining that difference could be a different "soil-to-interior dust relationship" in different areas. SAB Report § 3.5.2. The model presumes that the majority of a child's lead intake is from dust and that the importance of outdoor dust is in its contribution to interior soil. Therefore, the accuracy of the factor used to translate exterior soil levels to interior dust levels is absolutely crucial to the accuracy of any model predictions.

The Guidance Manual notes that although the default is presently set at 0.70, "[t]he user is cautioned, however, that the contribution of soil to dust concentration varies greatly from site to site, and site-specific soil and dust data should be collected for use in the model." Guidance Manual at 2.3.43. The

manual notes an extremely wide range of variation in the soil to dust ratio at sites studied by EPA:

> In sites where soil-to-dust coefficients have been measured **and where paint does not contribute greatly to dust,** the range was from 0.09 to 0.85. Among the sites where soil-to-dust coefficients have been measured are the following: East Helena, 0.85 (0.81 and 0.89); Midvale, 0.70 (0.68, 0.72); Butte, 0.26; and Kellog, 0.09. **Recent data suggest that the coefficient decreases over time at some sites where major sources of soil lead deposition are no longer active.**

Id (emphasis added). EPA's Technical Support Document supports a ratio of 28%, based on the EPA's January 1991 Draft of its Research and Development: Technical Support Document on Lead prepared for the Office of Solid Waste and Emergency Response. "Davis et al. (1990) measured the concentration of aluminum and silicon in soil and indoor dust and found the dust/soil ratios for both metals to be 0.28. Assuming that indoor aluminum and silicon are derived entirely from transport of soil into the house, these data support a conversion factor of 0.28 [Pb Dust = .28 x Pb soil]." Technical Support Document p. 3-22.[3/] Note, we do not intend to adopt the ratio of 0.28 as being correct. Instead, we question how EPA can support the use of 0.70.

Indeed, it is questionable that the EPA relies on a standard conversion calculation to estimate house dust when actual house dust data was available from the Study. This data demonstrates that interior dust does not bear a straight line relationship to soil levels. TRC points out that in many cases

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3/ Note that this document is a draft marked "do not cite or quote."

the actual dust leads exceeded outdoor soil leads, suggesting a large contribution to indoor dust from another source, most likely lead paint. TRC at 9. Indeed, if lead paint is the primary source of the house dust, then the modeling exercise itself would be invalid. If the predictions of interior dust from exterior soil are invalid, then remediation of the soil will not have the effect on blood leads asserted by the EPA. To the contrary, this dust data suggests that a focus on cleaning interior dust and cleaning up deteriorating lead paint would have a greater effect in reducing blood leads.

8. THE EPA HAS NOT USED THE BEST SCIENCE TO EVALUATE THE SOURCE OF THE LEAD FOUND IN THE SAMPLES

A limited study to speciate the lead found in the dust and soil samples should be conducted to assess with more precision and scientific certainty the true source of the lead which has been found in the surveys. There is good reason to believe that were such a study conducted, it would reveal that elevated lead levels in house dust are due primarily to deteriorating lead paint. The model presumes that the primary pathway to lead exposure for young children is house dust. Therefore, funds would better be spent on addressing lead paint abatement to diminish house dust levels rather than a proposal to dedicate millions of dollars to reduce soil levels already below the likely urban average to an unnecessarily and artificially low

number, while not addressing the true sources of the lead in the likely pathways of children.

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January 13, 1995

BY CERTIFIED MAIL
RETURN RECEIPT REQUESTED

Ms. Susan Pastor
Community Relations Coordinator
Office of Public Affairs (P-19J)
U.S. EPA, Region 5
77 West Jackson Boulevard
Chicago, Illinois 60604

Re: NL Industries/Taracorp Superfund Site
Granite City, Illinois
Comments on the October 1994 Proposed Plan

Dear Ms. Pastor:

This document is submitted for inclusion in the Administrative Record for the NLIndustries/Taracorp Superfund Site in Granite City, Illinois by AlliedSignal Inc., AT&T Corp., Exide Corporation, Gould, Inc., Johnson Controls, Inc., and NL Industries, Inc. (the "Parties"). The document summarizes and draws conclusions from the following documents, which, except for document number 6, are also attached:

1. The Granite City Lead Exposure Dataset: IEUBK Modeling and Evaluation of Soil Lead as a Risk Factor, by TRC Environmental Corporation ("TRC"), 1/6/95 (hereinafter "TRC Report");

2. NL Industries/Taracorp Site, Granite City, Illinois: Comments Addressing the USEPA's Use of the IEUBK Model to Justify 500 ppm/Pb Soil Clean-Up Level, by Morgan, Lewis & Bockius ("MLB"), 1/13/95 (hereinafter "MLB Comments");

3. NL Industries/Taracorp Site, Comments to Proposed Plan, by McLaren/Hart Environmental Engineering Corporation ("McLaren/Hart"), 1/12/95 (hereinafter "McLaren/Hart Comments on Proposed Plan"), including:

   a. Review of Public Record Documents for the NL Industries/Taracorp Site, by McLaren/Hart, 1/12/95 (hereinafter "McLaren/Hart Record Review"); and

b. Comments on the Madison County Lead Exposure Study and Related Documents, by McLaren/Hart, 1/12/95 (hereinafter "McLaren/Hart Comments on Exposure Study");

4. Comments on Exposure Study by Dr. Ellen J. O'Flaherty, 11/22/94 (hereinafter "O'Flaherty Comments");

5. Remedial Cost Analysis, Granite City Lead Site, by REACT Environmental Engineers ("REACT"), 1/12/95 (hereinafter "REACT Cost Analysis"); and

6. Summary Report, Evaluation of USACOE Remedial Action Program, Granite City, IL, by Earth Sciences Consultants, Inc. ("Earth Sciences"), 1/13/95 (hereinafter "Earth Sciences Report").[1]

Background

On March 30, 1990, the United States Environmental Protection Agency ("U.S. EPA") issued a Record of Decision for the NL Industries/Taracorp Superfund Site in Granite City, Illinois ("Site"), which required the cleanup of property once housing a secondary lead smelter that ceased operating in 1983, as well as surrounding commercial and residential property. Relying solely on a guidance document issued shortly before,[2] in its January 10, 1990 Proposed Plan for the Site, U.S. EPA set the residential soil cleanup level at 500 ppm lead in soil. Despite comments addressing U.S. EPA's illegal reliance on a guidance document for setting residential soil cleanup levels, the lack of evidence in the record supporting the level, and evidence supporting a significantly higher cleanup level, U.S. EPA maintained the residential soil cleanup level at 500 ppm in its Record of Decision. The Parties, now defendants in an action by the United States to enforce the terms of a November 27, 1990 administrative order, have offered to perform the cleanup required by the Record of Decision, but have declined to clean up to a level of less than 1,000 ppm lead in soil based on their review of the rationale presented in the Record of Decision and all extant scientific evidence.

At the time of the Record of Decision, no health study had been conducted in the area of the Site. As a result, no site-specific data existed on which to base the need for a cleanup. As part of their good faith offer to perform the cleanup required by the Record of Decision, the Parties also offered to perform a health study. While U.S. EPA refused to entertain the offer, it did commission the Agency for Toxic Substance and Disease Registry ("ATSDR") to perform a study similar to that proposed by the Parties. Nevertheless, U.S. EPA stated that it would not allow the results of the study to influence its choice of remedies at the Site. The following comments expressly request U.S.

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[1] The Earth Sciences Report is included with the City of Granite City's comments.

[2] The first U.S. EPA guidance on soil lead cleanup levels was entitled "Interim Guidance on Establishing Soil Lead Cleanup Levels at Superfund Sites (OSWER Dir. # 9355.4-02, 1989)." It has been superseded twice since its use at the Site, but U.S. EPA has not placed in the record to date any explanation addressing whether the newer guidances should result in a different soil cleanup level. We presume that the decision document entered after the close of the comment period will address the current guidance. Nevertheless, we continue to note that overreaching reliance on a guidance in rendering an administrative decision can rise to the level of illegal rulemaking if U.S. EPA fails to evaluate site-specific evidence in an even-handed manner. McClouth Steel Products Corp. v. Thomas, 838 F.2d 1317 (D.C. Cir. 1988).

EPA to reconsider its 1990 decision based not only on the information it refused to consider in 1990,[3] but also on the results of the "Madison County Lead Exposure Study, Granite City, Illinois" (hereinafter "Exposure Study") and what has been learned about the contribution of lead in soils to lead body burden in the interim. They further request U.S. EPA to realistically evaluate its use of the IEUBK model in reaching decisions about soil cleanup levels. Even when properly calibrated, it is only one tool for evaluating the potential health effects of lead contamination. Nevertheless, even the IEUBK model suggests in the present case that the massive soil removal demanded by U.S. EPA will not significantly affect blood lead levels.

Results of the Exposure Study

The final version of the Exposure Study has not been released. However, the Parties reviewed the draft Exposure Study during the ATSDR comment period and did not submit comments because they believed the study ultimately adequately addressed the status of the children in Granite City. The results of the Exposure Study indicate that any significant increase in childhood blood lead levels in the area of the Site are directly attributable to the age of the housing stock and the accompanying problems with lead-based pigments in interior and exterior paint. In fact, the blood lead levels in the community closely match those of a similarly situated community.

U.S. EPA did submit comments which took issue with many of the methods and conclusions summarized in the Exposure Study. The Parties have reviewed U.S. EPA's comments. The documents attached to this summary, as well as those produced by the Exposure Study's authors in response to the comments, indicate that the comments generally demonstrate either a lack of understanding of the study or a failure to understand the use of statistical and analytical tools as applied in the study. Unfortunately, they also indicate a preconceived notion that soil cleanup levels for lead should be less than 500 ppm, no matter what the scientific data developed across the nation at various lead sites may indicate and no matter what those scientists who have worked as public servants or scholars and have followed such issues for the better parts of their careers may say. For purposes of the following discussion, the Parties assert that the conclusions of the Exposure Study are essentially correct. The conclusions reached by the Exposure Study and the documents attached to these comments conclude that it is not only unnecessary from a health viewpoint to undertake the cleanup as envisioned by U.S. EPA, it is also a waste of time and money.

Summaries of Attached Documents

The documents attached to this letter are briefly summarized below. The documents should be consulted for more detail.

1. The TRC Report

The TRC Report analyzes the Exposure Study, the use of the IEUBK model to successfully account for the blood lead distribution found around the Site, and the U.S. EPA critique of the Exposure Study. The authors conclude that the blood lead levels found at the Site are related most strongly to housing condition. The housing condition influences blood lead levels to the extent that many of the older residences are coated with paints which utilized lead-based pigments. These paints contribute to house dust which, in turn, is ingested by children. Not surprisingly, the blood lead levels in the area are consistent with the recent data compiled in the NHANES III data set for similar communities, which also typically contain older housing stock subject to the same concerns.

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[3] The Parties to these comments expressly incorporate by reference their good faith offer dated August 31, 1990 and their comments to the administrative order dated December 20, 1990.

TRC's review of the IEUBK model demonstrates that Version 0.99d grossly overpredicts blood lead levels at soil levels in excess of 500 ppm. The overprediction can be explained and compensated for by adjustment of the absorption coefficient at appropriate levels of soil lead concentration. Calibration leads to a model run which replicates the Exposure Study data set. When the model is then used on the data set to replicate cleanup of houses included in the data set, it demonstrates that soil cleanup first will not result in an appreciable decrease in blood levels, second will not appreciably decrease the number of children with blood lead levels exceeding 10 μg/dl, and third can never reach U.S. EPA's stated goal of no more than 5% of the relevant population having blood lead levels in excess of 10 μg/dl. In fact, cleanup to 500 ppm rather than 1,000 ppm gains very little, despite the $14 million to $67 million extra expense identified in the enclosed REACT Cost Analysis.

Regarding U.S. EPA's critique, TRC questioned why U.S. EPA ran the IEUBK model without utilizing the site-specific data set for house dust available from the Exposure Study. Failure to use the data greatly skews the conclusions one can reach using the model and negates the conclusions drawn by U.S. EPA.

2. The MLB Comments

The MLB Comments conclude that the proposed 500 ppm cleanup standard is not appropriate for the Site for the following reasons. First, U.S. EPA has not demonstrated that the former smelter caused the soil lead levels at the Site because urban areas generally have higher soil lead levels anyway due to their history of heavy truck and automobile traffic (lead gasoline residue), older housing (lead paint), and various industries. Indeed, the Exposure Study demonstrates that soil lead levels and blood lead levels at the Site are typical of similar urban communities. In addition, U.S. EPA did not consider socioeconomic status and condition of housing even though both the Exposure Study and U.S. EPA guidance recognize the importance of these factors on blood lead levels. U.S. EPA also improperly discounted the beneficial effects of the follow-up counseling and education that was conducted as part of the Exposure Study.

The MLB Comments also conclude that, contrary to its own guidance, U.S. EPA improperly ignored the effects of lead paint in its application of the IEUBK model and, as a result, overpredicted the magnitude of the contribution from soil lead. In addition, the MLB comments question U.S. EPA's assumption regarding the effect of soil lead on dust lead. In assuming a soil to dust ratio of 0.70, U.S. EPA improperly ignored the effects of lead paint on dust lead.

3. The McLaren/Hart Comments on Proposed Plan, including the McLaren/Hart Record Review and the McLaren/Hart Comments on Exposure Study

McLaren/Hart analyzed the relevant documents in the Administrative Record as well as the Exposure Study, U.S. EPA's critiques of the Exposure Study, and the author's response to U.S. EPA's critique. McLaren/Hart concludes that the documents in the Administrative Record do not support the selection of a 500 ppm cleanup level for residential soils but rather support a cleanup level of 1,000 ppm or higher. Significantly, McLaren/Hart concluded that U.S. EPA did not properly take into account the potential for sources of lead other than soil, including paint. U.S. EPA also ignored other site-specific factors in choosing its cleanup standard at the Site.

McLaren/Hart also reviewed the Exposure Study and related documents. McLaren/Hart concluded that the Exposure Study was of high quality and the conclusions reached in the Exposure Study were supported by the data and the statistical analysis performed. McLaren/Hart also disagreed with the U.S. EPA critiques of the Exposure Study and concluded that, based on the Exposure Study: (1) the lead levels in children's blood in the Madison County study do not indicate an

imminent public health problem; (2) soil remediation is not likely to significantly reduce blood lead levels in children in general; and (3) soil remediation is not likely to significantly reduce blood lead levels in children with elevated levels of blood lead.

4. The O'Flaherty Comments

Dr. O'Flaherty reviewed the Exposure Study, the U.S. EPA critiques of the Exposure Study, and the author's response to those critiques.[4] Dr. O'Flaherty generally supported the quality of the Exposure Study as well as the conclusions in the Exposure Study and the statistical treatment of the data. However, Dr. O'Flaherty was particularly critical of U.S. EPA's critiques of the Exposure Study, stating that many of the criticisms by U.S. EPA miss the mark and do not seem connected to the section of the Exposure Study being commented upon. For example, Dr. O'Flaherty concluded that U.S. EPA commenters do not seem to understand that the Exposure Study design was not the conventional environmental epidemiology study design with an exposed community group and a control group.

In addition, Dr. O'Flaherty was extremely critical of the assessment of the Exposure Study conducted by A. H. Marcus. For example, Dr. O'Flaherty states that Dr. Marcus' conclusion that soil lead and dust lead are contributors to blood lead, based on similar patterns of decreasing concentration with increasing distance from the former smelter, is "absolutely unjustifiable" because such simple correlations cannot support such a conclusion. Dr. O'Flaherty concluded her comments by stating that the overall impression given by Dr. Marcus' reanalysis is that "the recommended soil remediation level was predetermined" and that the reanalysis is "superficial and careless, and bears little if any relationship to the data from the [Exposure] study."

5. The REACT Cost Analysis

The REACT Cost Analysis reviewed in detail the U.S. EPA cost estimates and the amounts allocated for the remediation of the residential areas at the Site. REACT also conducted an independent cost estimate for the residential cleanup for both a 500 ppm soil cleanup level and a 1,000 ppm soil cleanup level.

REACT's analysis included, among other things, a review of the Explanation of Significant Difference ("ESD") issued by U.S. EPA in January 1994 and the delivery orders related to the removal action that was planned for seventy (70) residences in August 1994. REACT noted that U.S. EPA has been very inconsistent with its use of its own cost estimates, constantly changing the average per residence cost. In addition, REACT concluded that, based on delivery orders issued to date, U.S. EPA was allocating approximately two to three times the amount of money per residence than was actually needed, due in part to interagency mark-ups of contractor fees. As a result, the residential soil cleanup under U.S. EPA's management would cost as much as $53 million more than if the Parties conducted the same cleanup ($82 million vs. $29 million). REACT also concluded that U.S. EPA's property characterization was flawed, creating the potential that entire properties would be remediated where only hot spots exist.

In addition, REACT concluded that the estimated cost difference between a 500 ppm level and a 1,000 ppm level ranges from $14 million to $67 million. The $14 million difference is based

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[4] Dr. O'Flaherty is an Associate Professor of Environmental Health and the Director of the Toxicology Training Program at the University of Cincinnati College of Medicine. Her curriculum vitae is included with her comments.

on a comparison of REACT's cost estimate for the 1,000 ppm standard ($15 million) to REACT's cost estimate for the 500 ppm standard ($29 million). The $67 million difference is based on a comparison of REACT's cost estimate for the 1,000 ppm standard ($15 million) to U.S. EPA's cost figures for the 500 ppm standard ($82 million). As noted above and in the other attachments, no statistically significant benefit in the protection of human health would result from the expenditure of this extra $14 million to $67 million.

6. The Earth Sciences Report

The Earth Sciences Report analyzes the removal action that was conducted by U.S. EPA, through the U.S. Army Corps of Engineers ("USACOE"), in August 1994 at certain residences in and around Granite City.[5] The original removal action was planned for approximately seventy (70) residences; however, only a few residences were completed as a result of the lawsuit filed by the City. See footnote 5.

The Earth Sciences Report identifies actual problems that were witnessed by Earth Sciences during the removal action. For example, the report concluded that air monitoring conducted by USACOE was inadequate for determining the lead levels to which on-site workers and nearby residents may have been exposed. In addition, the review of the work by Earth Sciences revealed the following problems during the removal action: (1) inadequate site security, including the presence of children at and around residences that were being cleaned; (2) cross-contamination of clean areas outside the excavation zone; (3) recontamination of the residences being cleaned up; and (4) damage to the City's infrastructure, including damage to sidewalks. The Earth Sciences Report is significant because it reveals the actual problems that will occur if the U.S. EPA Proposed Plan is implemented.

Choice of Residential Cleanup Level

In their good faith offer in 1990, the Parties committed to a compromise cleanup standard of 1,000 ppm lead in soil. The Parties continue to believe that the evidence, both in general and that specific to the Site, supports a considerably higher level. Evaluation of any cleanup remedy must be consistent with the National Contingency Plan, rules promulgated by the agency pursuant to CERCLA. The nine factors cited in the NCP for evaluation are: (1) overall protection of human health and the environment; (2) compliance with ARARs; (3) long term effectiveness and permanence; (4) reduction of toxicity, mobility, or volume through treatment; (5) short-term effectiveness; (6) implementability; (7) cost; (8) state acceptance; and (9) community acceptance. The Illinois Environmental Protection Agency has not spoken recently on the matter of Site cleanup. We are aware that the agency did concur with the remedy five years ago. We do not know what the agency's position is today. Consequently, the Parties limit their analysis to the remaining eight factors.

1. Threshold criteria: overall protection of human health and the environment; and compliance with ARARs

As noted above, a 1,000 ppm cleanup standard is equally as protective of human health and the environment as a 500 ppm standard. The Exposure Study and the attached reports reveal

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[5] The City of Granite City filed suit against U.S. EPA shortly after the removal action began and requested a temporary restraining order ("TRO") to halt the cleanup. U.S. EPA voluntarily agreed to stop the removal action until the hearing before the Court on the TRO. The parties reached a settlement at the hearing which generally stated that U.S. EPA would only conduct residential soil removal at a limited number of residences, and the City and the Defendants would be allowed to conduct a recontamination study on those residences.

that lowering the cleanup standard from 1,000 ppm to 500 ppm would not result in a statistically significant reduction in blood lead levels. In addition, a 1,000 ppm cleanup standard complies with ARARs to the same extent as a 500 ppm cleanup standard. For example, Illinois Department of Public Health ("IDPH") regulations use a level of 1,000 ppm for the permissible limit of lead in soil which is readily accessible to children. 77 Ill. Admin. Code sec. 845.50(b). IDPH actually increased the level from 200 ppm to the current 1,000 ppm level in February 1993, realizing that 1,000 ppm was sufficiently protective of human health and the environment.

2. **Primary balancing criteria: long-term effectiveness and permanence; reduction of toxicity, mobility, or volume through treatment; short-term effectiveness; implementability; and cost**

The long-term effectiveness and permanence criteria as well as the reduction of toxicity, mobility, or volume through treatment criteria would be satisfied to the same extent for a 1,000 ppm cleanup standard as it would for a 500 ppm standard. In addition, the short-term effectiveness and implementability criterion actually favor implementation of a 1,000 ppm standard instead of a 500 ppm standard. As noted above, the Earth Sciences Report reveals the serious short-term effectiveness and implementation problems associated with large-scale residential soil removal, including site security, cross-contamination, recontamination, and infrastructure damage. Furthermore, we understand that the City will be submitting comments amplifying the short-term effectiveness and implementation problems associated with residential soil removal, including traffic-related problems and the negative economic impact of the proposed cleanup.

In addition, the cost criteria in the NCP supports the 1,000 ppm standard over the 500 ppm standard. As noted above and in the REACT Cost Analysis, the cost difference between a 500 ppm standard and a 1,000 ppm standard ranges from $14 million to $67 million. This is an extraordinary amount given that no statistically significant reduction in blood lead levels would occur if a 500 ppm standard were chosen over a 1,000 ppm standard. CERCLA and the NCP mandate that U.S. EPA consider cost in selecting a Superfund remedy. Selecting a remedy that costs $14 million to $67 million more than an equally protective remedy would be a clear violation of this mandate.

3. **Modifying criteria: community acceptance**

Through two separate administrations, the City of Granite City has consistently voiced its objection to the 500 ppm proposed cleanup standard. For example, when U.S. EPA filed its lawsuit against the Parties, the City intervened as Intervenor-Defendants. In addition, when U.S. EPA began a removal action on certain residences in August 1994, the City sued U.S. EPA to stop the removal action. We also understand that the City will be submitting a separate set of comments today objecting to the 500 ppm cleanup level in the Proposed Plan. U.S. EPA quite obviously does not have community acceptance of its proposed remedy.

Conclusion

In sum, the Parties do not believe that the 500 ppm residential soil cleanup level as chosen by U.S. EPA in the Proposed Plan is appropriate for the Site. U.S. EPA's selection of the 500 ppm level as the final residential soil cleanup standard would violate the NCP and constitute arbitrary and capricious conduct. The Parties believe that a much higher standard would protect both human health and the environment and save at least $14 million to $67 million.

Sincerely,

Louis F. Bonacorsi

Joseph G. Nassif

Dennis P. Reis

Attachments

cc: John H. Grady, Esq.

0012200.01 January 13, 1995